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A PRACTICAL CONCEPT FOR THE TREATMENT OF MAJOR AND MINOR BURNS

THE IMPORTANCE OF TIMING THEREIN

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THE MANAGEMENT OF THE VICTIM of severe burns has always presented one of the major problems of surgical experience. During, and since the first phase of the World War I, 1914-1918, many surgeons and investigators devoted much time and effort to a study of the principles and details of such management. These investigations have had for their aim the preservation of life and return to useful citizenship of the victims of such misfortunes with as little loss of time as possible. That the attainment of either of these desiderata is not easy or certain is attested to by the fact that there is still no unanimity of opinion as to the best method of local treatment.

The present conflict has hitherto been characterized by an enormous increase in burned persons. These cases have been the result of an aerial bombardment of civilians; fire in the cockpit or fuselage of aircraft; oil and flash burns on shipboard, both mercantile and naval, and as the result of the general employment by all combatants of tanks. It is probable, moreover, that in the very near future flame throwers and the employment of corrosive gases will present further problems.

In an attempt to elucidate the problems indicated above, there have appeared, more especially since the present phase of the war began, many publications by proponents and protagonists of several essentially different techniques; some new and some previously proven methods. Among these different methods the more important are probably tannic acid with or without silver nitrate as an adjuvant; triple dye jelly or solution; normal sodium chloride solution and/or hypochlorite employed as bath, compress or bag (Bunyan).[13] Needless to say, the introduction of sulfonamides has been tested by numerous surgeons and experimental workers. It would appear that in the hands of those thoroughly trained in the individual techniques each of these methods may be used with profit.

It is not our intention in this communication to add to the confusion which, to a certain extent, envelops the subject. It is our purpose, however, in a

modest fashion, to outline a method which, in our hands, has proven itself to be satisfactory, simple and safe. This definite plan of treatment is comprehensive enough to include the use of any well recognized and accepted therapeutic principle.

We wish, at the outset, to emphasize the necessity for having such a plan for the treatment of major burns; whether in the armed services, or in civil industrial practice, especially in the emergent situation of today. The plan we have adopted at the Montreal General Hospital is based on "timing" as understood in all modern treatment. This is not a new principle. Perhaps it has hardly been emphasized enough in scientific medical teaching. We submit that, for the successful practice of surgery today, constant practical application of accurate timing in treatment is essential. It is as necessary in the medical world as in all phases of modern life whether in war or peace. Our plan is outlined in some detail in the accompanying illustrations, charts, and tables. It has been employed successfully at the hospital for several months.

The history of modern burn treatment may be roughly divided into four consecutive periods; each merging into the other; each marking an advance in knowledge; all resulting in progressive and continuous improvement in mortality percentage; and in local functional and aesthetic results. These deductions are well shown in the statistics from leading clinics. The periods referred to are:

(1) That previous to the adoption of tannic eschar treatment.

(2) The period marked by the adoption of the principle of tannic escar treatment as suggested by Davidson (1925).[16]

(3) The period following this, marked by full recognition of the rôle of shock,[8, 9, 14, 15, 20, 21, 27, 28, 34, 35] and the gradual development of means to combat it; in particular the controlled use of blood substitutes. This period actually began much earlier but has only been generally recognized during the last decade.

(4) The period subsequent to the introduction of sulfonamide therapy for topical application—about 1940.

The improvement during these periods was well shown by Farmer,[18] in a recent paper read before the American Association of Surgery of Trauma in Boston, with percentages of 35–16–11 and 2.5 per cent for the corresponding periods at the Sick Children's Hospital, Toronto, Canada. The improvement in morbidity and functional results is just as striking. On this side of the problem certain details of local treatment have played important rôles, i.e., the importance of early skin grafting, and the abandonment of restrictive eschar treatment on hands, fingers, etc.

When and where the above facts are fully realized, the surgeon will see that any attempt to substitute or revise any phase of burn treatment must take full cognizance of these factors. In full recognition of this, we have planned our treatment to include the basic principles above mentioned, and timed the plan to *anticipate* each of the problems commonly met with in turn, namely: (1) Shock, during the first 24–48 hours, (2) Toxemia, from the second to

the fifth day. (3) Sepsis during the subsequent week or 10 days. (4) Finally, scarring and contractures. (See Table III for details)

At this point, it is necessary to refer to several features of the burn problem which have such direct bearing on results that they may influence the surgeons' treatment:

(1) The extent of surface area of the burn—In this connection, the recent tendency to divide the burns into *major* and *minor* problems, as encouraged in Britain particularly, has helped to simplify matters. (Berkow's Tables[6] are used in our surface estimations)

(2) Important as the foregoing is, it must be modified or qualified, at once, by reference to the agent responsible for the burn, such as actual fire, hot liquid, electricity, chemicals, radiant heat, hot metals, etc. In this connection the length of exposure to the burning agent must also be considered. These comments require no elaboration to the initiated.

(3) The well known comparative severity of burns in children.

(4) The elapsed time between the burn and treatment. In major burns this may be extremely important as regards shock, and 30 minutes may be said to be a long time under the best conditions. Minor burns, on the other hand, frequently are neglected for several days, when infection and its consequences may change them into more serious problems.

(5) Other factors contributing to shock, such as exposure to the elements, fatigue, exhaustion, lack of food and water, etc., particularly in the fighting services.

(6) Concomitant injuries may be severe and greatly complicate treatment. Burns in the air force or air accident crashes frequently present very serious and even fatal injuries of other kinds.

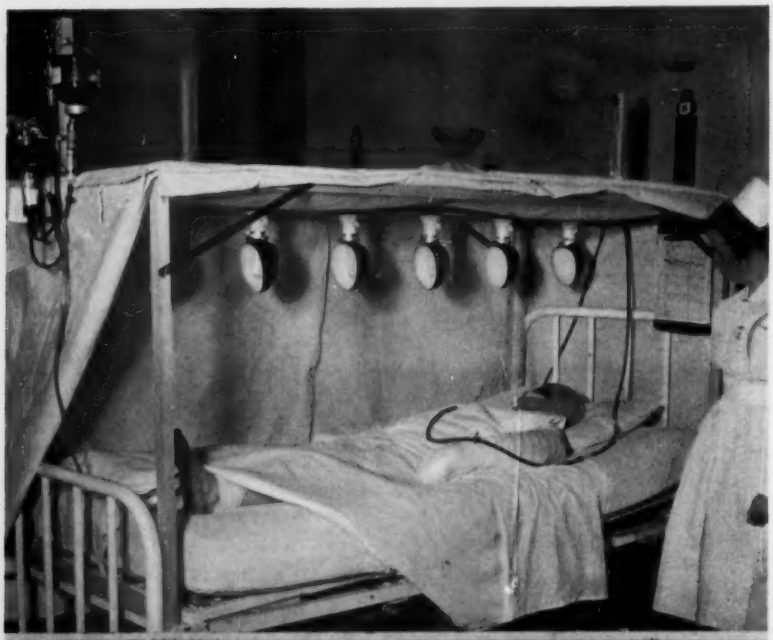
(7) The type and extent of first-aid care, particularly as regards exclusion of air and maintenance of body warmth.

(8) Finally, with regard to the depth of the burn and the various classifications used to describe this. The writers believe that the only decision needed is whether skin-grafting will be necessary or not. Therefore, as it is difficult, if not impossible to gauge the depth accurately in the early stages, the arguments regarding different classifications seem superfluous. Wakeley[31] has pointed out that five days is the minimum for recognition of depth.

PROCEDURE IN MAJOR BURNS OF THERMAL TYPE

Having regard for the above facts and previously mentioned sequence of complications to be anticipated, the timed schedule of treatment is carried out as in Table III. To do this most effectively requires careful prearranged organization of personnel, equipment, and supplies.

Regarding personnel, it has been found best to have separate burn and shock teams, each composed of one attending surgeon, one resident and interns,



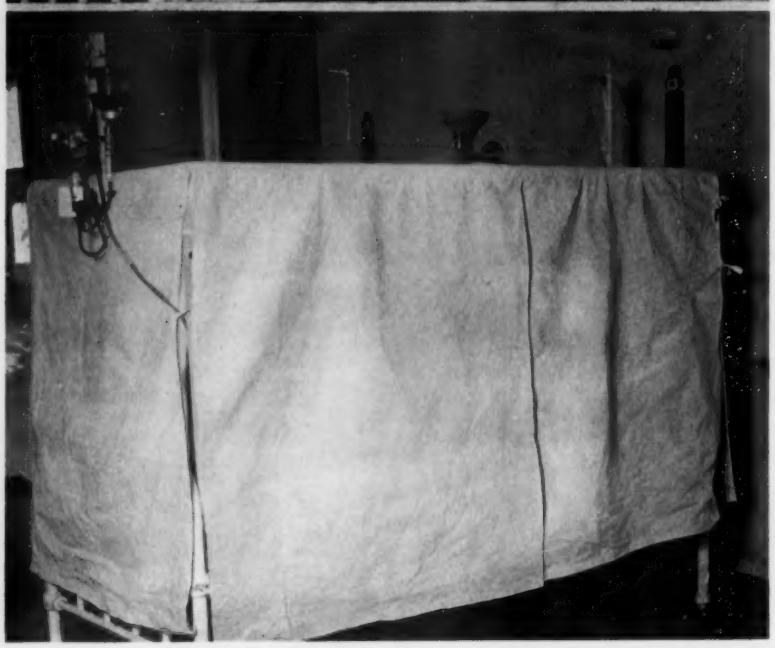


FIG. 2.

FIG. 1.—Showing burn tent opened.
FIG. 2.—Showing burn tent closed.

all operating under a senior surgeon. Trained graduate nurses and orderlies are also part of our set-up. Equipment includes special burn tents shown in Figures 1 and 2. Supplies should include an adequate amount of stored plasma or serum, tannic acid, silver nitrate, 5 per cent sulfathiazole emulsion, and 4″ x 6″ cuts of 1 or 2 Mm. curtain-mesh.

On admission, the major burns are placed at once in the burn tent, and the temperature gradually, not rapidly, raised to 80° F. The shock team functions first, estimating the hemoconcentration, and immediately administering plasma diluted 50 per cent by saline. This is done by a continuous drip in a cut-down intravenous, *via* the long saphenous vein at the ankle. A cannula, and not a needle, is used as a routine. It may be necessary to milk the plasma into the vein at the start. We have evolved a special double roller for this purpose.

When the above measures have been instituted, usually about one hour after admission, local treatment is begun. Operating room technique is used in the ward, in the tent bed, and only very rarely is anesthesia employed. Morphine gr. 1/4 is administered on admission and repeated during the first hour if necessary (synergistic effect). For large surface areas (trunk or thigh), when life-saving is involved, tannic acid–silver nitrate eschar[7, 16] is used. So far, we have not found a better type of eschar. In all critical areas, in moderate areas otherwise, and for all minor burns, we are using a thick pressure-type dressing.[2, 3, 4, 24] A mask is used for the face. We have modified the dressing by using a basic dressing of 5 per cent sulfathiazole oil in water emulsion (M. G. H. formula), as described in detail by one of us (Ackman)[1, 2] in 1942, and has been found to be a highly successful method of preventing infection, or controlling it in delayed, contaminated, or actually infected cases. The details of the development and preparation of the emulsion, as well as our experience with it in burn therapy, and other surgical practice, have been described in the literature this year.[1, 2]

The topical use of sulfonamides in crystalline form, in powder, paste, suspension or emulsion, is already well established, so much so that to eliminate this form of treatment in burns would be a backward step. Before applying an eschar it is possible to powder a burned surface, and similar treatment may be used at other times. In critical areas, for all practical purposes, we have found the M. G. H. emulsion offers a particularly satisfactory adjunct to pressure dressings. The bacteriostatic action prevents or controls infection and, because of this, the frequency of the redressing may be timed. It is usually possible to leave the original dressing or for at least one week without discomfort and with little odor, more often none. Moreover, this emulsion dressing affords a single continuous form of treatment from first-aid to final healing, even with grafting.

Local tissue concentrations of sulfathiazole are surprisingly high, 120 mg. + per cent in 24 hours; blood levels are low and fleeting (maximum 3.5 in one hour) even with large amounts (10 ounces) of the emulsion. We recognize, as have others, the anesthetic action of the sulfonamide, akin to the effect of benzocaine.

It is interesting to note that when primary débridement is complete the emulsion dressings may be left in place for as long as two weeks without disadvantage. On the other hand, should the burned area require some revision or inspection during the first few days, as when progressive skin separation occurs, redressing is comparatively painless and free from bleeding. The use of the basic "sulfamesh" is an important factor, which permits inspection of the burned area at any time, and it need not be changed. The sulfathiazole emulsion dressing, with its strong bacteriostatic action, materially assists early healing of superficial burns (6–7 days). In deeper burns, it permits early skin grafting and thus hastens the timing schedule.

We find the use of the emulsion facilitates the softening and separation of slough in severe cases. It is not necessary in these cases to completely replace and renew the basic "sulfamesh" dressing. Generally speaking we find it quite sufficient to supply additional emulsion on the renewed gauze layer.

The M. G. H. emulsion, we believe, has distinct advantages over any other medium hitherto presented. It is simple and clean to handle. It offers economy in nursing and medical attention, and in the materials required. Its constituents are readily available and inexpensive. Sterility tests are negative, while its stability at extremes of temperature has been proven. The anesthetic effect is noteworthy.

EMULSION SULFATHIAZOLE 5% (M. G. H. FORMULA)

Sulfathiazole (finely powdered)	5%
Triethanolamine	2%
Distilled water	24%
White beeswax	5%
Liquid paraffin	64%

TECHNIQUE

Step-by-Step Procedure

1. Treat shock first. (See Table III for details)

2. With aseptic technique, cleanse burned area with soapsuds made from castille soap—without anesthesia.

3. Wash with saline.

4. Apply single layer of 4″ x 6″ "sulfamesh" strips* (1–2 Mm. mesh), prepared beforehand or at time of treatment. Individualize fingers, *etc.* (This basic dressing may be left on at redressings). Mask is used for the face.

5. Apply over this ordinary gauze dressings of 3—4 thicknesses, generously impregnated with emulsion. Do not individualize fingers, *etc.* (Fig. 3).

6. Pressure dressing, with cotton waste outside this. Do not individualize fingers, *etc.*

7. Wrap in sterile towel or towels.

8. Bandage firmly, with 4″–6″ flannel or flannelette bandages cut on the *bias*,† or dressing gauze roller bandages 4–6 layers in thickness, or ordinary roller bandage if the former are not available (Fig. 4).

* Cut from curtain material of 1 or 2 Mm. mesh. In Canada called English curtain-mesh. May be obtained from T. Eaton Co. Ltd.

† Suggested by Dr. J. Carl Sutton of our surgical staff.

9. A plaster moulded splint is added for hand, forearm, *etc.*, in the functional position (Fig. 5).

Steps 6, 7, 8 and 9 minimize contamination. Note: In redressing, at whatever date, the "sulfamesh" is not changed unless débridement is indicated. This technique reduces pain and bleeding to a minimum, and permits removal of outer dressings to be performed more easily.

FIG. 3.

FIG. 4.

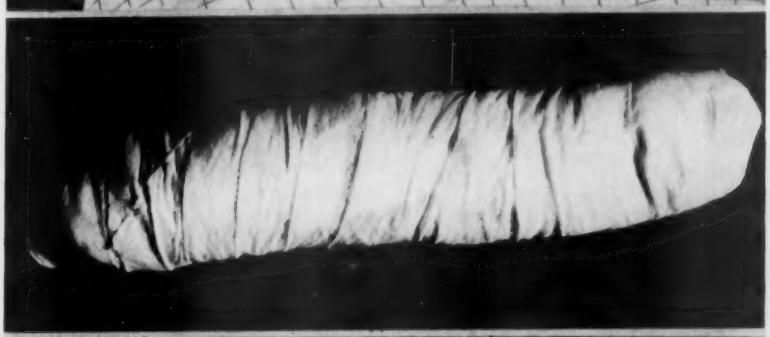


FIG. 5.

FIG. 3.—Showing basic "sulfamesh" dressing.
FIG. 4.—Showing completed burn dressing.
FIG. 5.—Showing completed burn dressing with posterior plaster mould.

TABLE I

SUMMARY OF DATA OF 120 CASES OF BURNS TREATED

Number of major burn cases	31
Number of minor burn cases	89
Total number of burn cases	120
Thirty-one major burns as above—2nd°* only	19
Mixed 2nd° and 3rd°	12
Healing time of 2nd° burns	Longest 18 days
	Shortest 6 days
	Average 7—10 days
Healing time of 3rd° burns	Longest 66 days
(Including skin grafting)	Shortest 21 days
Number of skin grafts (split grafts)	15
(100% "take" in all cases).	

INFECTIONS

General sepsis after treatment	None
Local sepsis after treatment	None

Pretreatment infections resulting from delayed admission—15. All controlled by first dressing. (One only, *Staph. pyogenes hemolyticus*, with some difficulty. Sulfathiazole-resistant type)[37].

COMPLICATIONS

Deaths	None
Number of sulfonamide rashes	Two
(Corrected by switch to 10% sulfanilamide emulsion)	
Number of toxic fevers	None

* Classification used—three degrees only.

TABLE II

LABORATORY INVESTIGATIONS SHOWING THE CONCENTRATION OF SULFATHIAZOLE IN LOCAL TISSUES TREATED WITH THE M. G. H. EMULSION

Dressings not Changed

Elapsed Time after Treatment—Average of Estimations in Mg. Per Cent

(Total Sulfathiazole Only)

Clinical Cases	M. G. H. Laboratory	Independent Laboratory*
24 hours	120 mg. per cent	124 mg. per cent
48 hours	78 mg. per cent	76 mg. per cent
72 hours	45 mg. per cent	43 mg. per cent
4 days	34 mg. per cent	—
5 days	30 mg. per cent	—
7 days	28 mg. per cent	—
Laboratory Animals	**M. G. H. Laboratory**	**Independent Laboratory***
24 hours	122 mg. per cent	129 mg. per cent
48 hours	—	65 mg. per cent

BLOOD LEVELS OF SULFATHIAZOLE IN LOCAL TISSUES TREATED WITH THE M. G. H. EMULSION

Dressings not Changed

Clinical Cases	M. G. H. Laboratory	Independent Laboratory*
1 hour	Total 3.5 mg. per cent	—
2 hours	3.1 mg. per cent	—
3 hours	3.0 mg. per cent	—
12 hours	1.3 mg. per cent	—
24 hours	None to trace only	—
Laboratory Animals	—	—
1 hour	—	12.1 mg. per cent
2 hours	—	7.6 mg. per cent
3 hours	—	3.5 mg. per cent total
4 hours	—	1.5 mg. per cent
5 hours	—	Trace only
6 hours	—	None

* These independent laboratory experiments were maintained through the courtesy, cooperation and material assistance of Messrs. Charles E. Frosst & Company, Montreal, Canada.

LABORATORY INVESTIGATIONS OF THE EMULSION

Molecular solubility of sulfathiazole in the emulsion, as checked in both M. G. H. and an independent laboratory —5.5% (in water phase).

Compared with molecular solubility in a mixture of water and 6% triethanolamine alone — 3.9%.

Compared with molecular solubility in water alone 0.07%.

It will be seen that sulfathiazole in the emulsion has a molecular solubility 800 times greater than in water, due, by inference, to the triethanolamine. The experiment indicates that 2% triethanolamine dissolved in the water phase of the emulsion holds a molecular solution of sulfathiazole in the emulsion of over 5%. Since the emulsion contains 24% of watery phase, and this watery phase holds 5.5% of sulfathiazole, then the total sulfathiazole in solution in 100 Gm. of the emulsion is 24 x 5.5 = 1.32 Gm., which, in turn, represents 26% of the total sulfathiazole present in the emulsion.

From the above experiments it will be seen that the 5% M. G. H. emulsion, while allowing for a remarkably high molecular concentration of sulfathiazole, actually has a very satisfactory and prolonged curve of delivery into the adjoining tissues. These experimental and clinical results indicate the superiority of an emulsion base over ordinary ointment bases.

GRAFTING OF BURNED AREAS

How Organized Timing Permits Early Skin Grafting

Modern burn treatment aims at an assessment of the burn damage as soon as shock and toxemia have passed, and the immediate start of reconstructive procedures. Tannic acid eschar and effective shock treatment have lowered burn mortality; now, early skin grafting is reducing burn morbidity. The patient is no longer allowed to suppurate in granulations and eschars for weeks and months while poor and useless skin is coaxed from burn margins, sweat glands, and hair follicles. The burn wounds are surfaced with the patient's own skin as soon as feasible. With these thoughts in mind, efforts have been directed in the Montreal General Hospital, toward the early preparation of burned areas for grafting.

By early grafting we mean grafting as close to the burn-time as the patient's condition will permit. Theoretically, and from the skin grafting viewpoint, this would be about the 6th or 7th day, when the postburn perivascular edema in the subepidermal and subcutaneous layers is subsiding. Practically, this conflicts with the toxemic phase, and we are usually unable to apply our grafts, in burns of any extent, until the 14th day onward. However, we feel there is an optimum time for grafting, and that it is early, before granulations have formed to any extent. As time goes on the granulations increase, grafting is less certain, and the resultant subgraft scar detracts from the final result.

TABLE

TIMING IN THE HOSPITAL TRE

THE MONTREAL GE

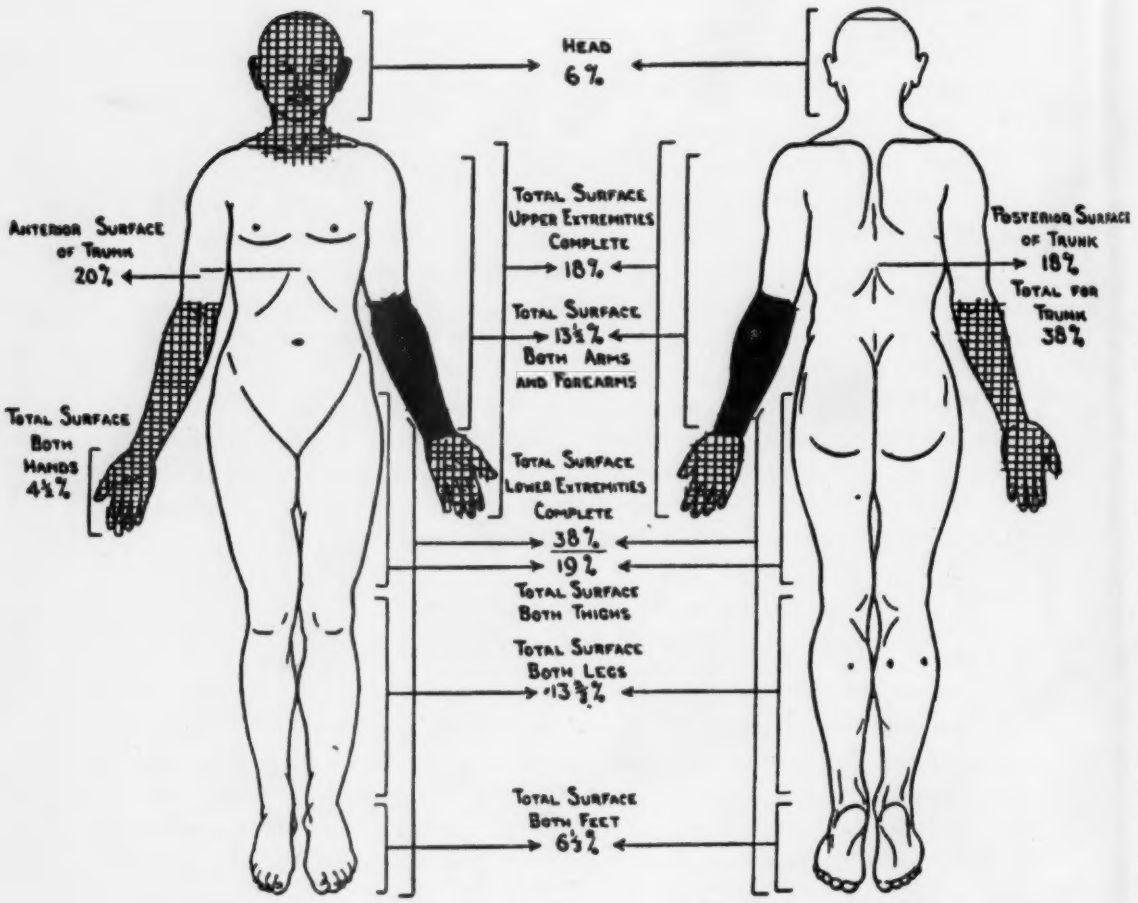
		PERIOD OF SHOCK 48 HOURS	
	FIRST HOUR	FIRST 24 HOURS	SECOND 24 HOURS
GENERAL TREATMENT	HYPO MORPHINE 1/4 GR. REPEAT IN THE HOUR	CONTINUE MORPHINE AS MAY BE INDICATED DURING THIS PERIOD	
SHOCK TEAM 24-HR. SERVICE		CORTIN 25 CC. IN EXTREME CASES	CONTINUE CORTIN INTRA MUSCULARLY 10 CC. Q. 6 H AS INDICATED FOR 3 OR DAYS
UNDER SUPERVISION SENIOR SURGEON	SERUM OR PLASMA IN AMTS. OF 500 CC.	CONTINUE TO A MINIMUM OF 2,000 CC. (PLASMA OR SERUM) FOR 10% BURN IN 48 HRS. CONTINUOUS INTRAVENOUS DRIP CONTROL THE AMOUNT OF SALINE AND SERUM OR PLASMA BY HEMOGLOBIN READINGS.	
	HEAT — DEVELOP SLOWLY TO 80° UNDER TENT		A.T.S. 1,500 UNITS
	WATER BY MOUTH CONTAINING CARBOHYDRATE FOR FIRST 24 HOURS		FLUIDS AS TOLERATED
	OXYGEN ENDOPHARYNGEAL TUBE		
	INTRAVENOUS GLUCOSE SALINE IF NEEDED		ENEMA
LOCAL TREATMENT	DELAYED UNTIL INTRAVENOUS SHOCK TREATMENT BEGUN AND EFFECTIVE	DÉBRIDEMENT WITHOUT ANESTHETIC	
BURN TEAM		TRUNK, THIGHS, LEGS, FEET, ARMS, FOREARMS SPRAY WITH 10% TANNIC ACID FOLLOWED BY 10% SILVER NITRATE. REPEAT Q. 1/2 H. UNTIL TANNED	
UNDER SUPERVISION OF SENIOR SURGEON		CRITICAL AREAS—SULFATHIAZOLE EMULSION DRESSINGS	DRESS AND DÉBRIDE ON 2ND DAY ONLY IF INDICATED
LABORATORY EXAMINATIONS	HEMOGLOBIN ESTIMATION OR HEMATOCRIT OR R.B.C. COUNT	REPEAT Q. 1 H UNTIL HEMOCONCENTRATION CONTROLLED, AND THEREAFTER AT LEAST Q. 4 H.	BLOOD CHEMISTRY TOTAL PROTEIN (ALB. AND GLOB.) CHLORIDES SUGAR CO_2 COMB. POWER
		CULTURE OF BURNED SURFACE (BEFORE TREATMENT)	CULTURES WITH DRESSINGS URINALYSIS
CLINICAL DATA	ADMISSION T.P.R.	CHART PULSE Q. 1 H. B.P. Q. 1 H. HEMOGLOBIN % Q. 1 H.— 4 HRS. THEN Q. 4 H. TEMP. Q. 4 H.	T.P.R. Q. 4 H. B.P. AND P. CHART Q. 1 H.
CHART		INTAKE AND OUTPUT DAILY PLASMA OR SERUM SALINE OR GLUCOSE SOL. BLOOD CHEMISTRY CORTIN	INTAKE AND OUTPUT DAILY PROGRESS NOTES

TABLE III

HOSPITAL TREATMENT OF MAJOR BURNS

IONTREAL GENERAL HOSPITAL

	TOXEMIA	GRANULATION & INFECTION	HEALING
24 HOURS	2ND TO 5TH DAY	5TH TO 14TH DAY	2ND TO 6TH WEEK
DURING THIS	SEDATIVES MORPHINE OR NEMBUTAL, ETC., AS INDICATED	SAME	SAME
ORTIN INTRA-10 CC. Q. 6 H. FOR 3 OR 4	SULFONAMIDES INTRAVENOUS OR BY MOUTH ONLY IF URINE OUTPUT 1,000 CC. 1 GRAM Q. 4 H.	SAME	SAME AS INDICATED
A OR SERUM) VENOUS DRIP. OR PLASMA BY	BLOOD SUBSTITUTES AND/OR INTRAVENOUS SALINE AND GLUCOSE AS INDICATED BY BLOOD CHEMISTRY	TRANSFUSIONS 500 CC. Q. 2 DAYS AT LEAST DEPENDING ON R.B.C.	
NITS	HEAT CONTINUED THROUGHOUT THIS PERIOD	HEAT MAY BE DISCONTINUED	
LERATED	FLUIDS MINIMUM OF 3,000 CC. BY MOUTH	SAME	FLUIDS 2,000 CC.
	DIET HIGH PROTEIN AND CARBOHYDRATE	SAME DIET	SAME DIET
	ENEMA Q. 2 DAYS	VITAMIN TABLETS CATHARTICS, AS INDICATED	VITAMIN TABLETS CATHARTICS, AS INDICATED
	UNROOF ANY INFECTION AND APPLY SULFATHIAZOLE EMULSION DRESSING	UNROOF ANY INFECTION AND APPLY SULFATHIAZOLE EMULSION DRESSING	UNROOF COMPLETELY AT START OF THIS PERIOD—2ND DEGREE BURNS REDRESS WITH SULFATHIAZOLE EMULSION AS NECESSARY
BRIDE ON 2ND NDICATED	CRITICAL AREAS DRESS AND DÉBRIDE Q. 2 DAYS ONLY IF INDICATED. DECISION BY SENIOR SURGEON	DRESSING DECISION BY SENIOR SURGEON	3RD DEGREE BURNS ALL AREAS SALINE DRESSINGS OR SULFATHIAZOLE EMULSION UNTIL CLEAN, THEN GRAFT AT ONCE.
TRY	BLOOD CHEMISTRY	SAME	IF INDICATED
N (ALB. AND	SUGAR		SULFONAMIDE LEVEL
	UREA AND CREATININE		
	SULFONAMIDE LEVEL		
VER	BLOOD COUNTS & HB.	SAME	BLOOD COUNTS & HB.
H DRESSINGS	CULTURES WITH DRESSINGS	SAME	SAME
	URINALYSIS DAILY	URINALYSIS (ONE)	SAME (WEEKLY)
	T.P.R. Q. 4 H.	SAME	SAME
IART	B.P. AND P. CHART AS INDICATED	DISCONTINUE	
UTPUT DAILY	INTAKE AND OUTPUT DAILY	SAME	SAME
	CHART	SAME	SAME
	PLASMA OR SERUM		
	SALINE		
	GLUCOSE		
	SULFONAMIDES	NOTES ON GRANULATION	NOTES ON GRANULATION
ES	PROGRESS NOTES		OPERATION NOTES



CHILDREN

Lower Extremities	38 − twice (12 — age)
Trunk	38 + half (12 — age)
Head	6 + (12 — age)
Upper Extremities	18 + half (12 — age)

Degree of Burn

1st Erythema

2nd Partial destruction

3rd Complete destruction

ESTIMATION OF TOTAL AREA BURNED 20% OF BODY

Summary:

Face—all, 2nd degree
Left ear—possibly 3rd degree
Forearms } Right, all 2nd degree
Wrists }
Hands—all 2nd degree } Left, largely 3rd degree

CHART 1A (See Chart I opposite)

From a reconstructive point of view, any burn dressing that permits easy removal and inspection at the end of 6, 8 or 10 days, or even earlier, is the ideal dressing. This is especially so on the critical areas, such as face and flexor surfaces, where contractures are so liable to ensue. Saline baths and dressings permit such inspection from the first, but, in our experience, have been cumbersome and impractical. The eschars, at present in use, do not permit such inspection. To date, sulfathiazole emulsion dressings of these critical areas have proven eminently satisfactory and the foregoing wide-mesh ("sulfamesh") gauze technique has been worked out. Contrary to the opinions of Blair and Brown,[10] and Brown and McDowell,[11, 12] that a wide-mesh gauze is a painful dressing and permits granulations to grow up through the mesh, we have not found it so, and feel the wide-mesh strands provide a mechanical factor, "splinting" or "snubbing" the wound surface, providing a more secure dressing, with better drainage, and is just as easily and painlessly removed as is fine-mesh gauze.

The burned areas are "papered" with 4 x 6 inch sheets of "sulfamesh." This clings snugly to the wound surface. A liberal coating of sulfathiazole emulsion is applied and dressing built up with gauze-cuts and cotton waste, as recommended by Brown and McDowell,[12] for a pressure dressing. A few days of such treatment, with change of dressings at three-day or longer intervals, and the area is ready for grafting. Earlier in our experience dressings were changed to saline soaks 24 hours before grafting, fearing the emulsion might interfere with the "take." Apparently this is not so, for the emulsion dressing is now removed in the operating room and the graft applied without further preparations, notwithstanding a fairly heavy sulfathiazole content of the underlying tissues.

Our grafts are of the split variety, half or three-quarter thickness, cut with the Padgett dermatome. These are transferred to "sulfamesh" gauze and sewn in with the gauze. The wide-mesh gauze, again, provides an excellent mechanical factor, and even more so with the thin layer of cement still adherent on the skin from the dermatome. With the aid of a basting suture it "snubs" the graft well into position, with little chance of movement. The dressing is built up with wisps of absorbent cotton wrung out of sulfathiazole emulsion, packed tightly against the "sulfamesh" and covered over with the gauze-cuts and cotton waste.

Split grafts, with this technique, have been uniformly satisfactory. In our hands, the "pinch graft" has been relegated to the sidelines, except where insufficient normal skin exists.

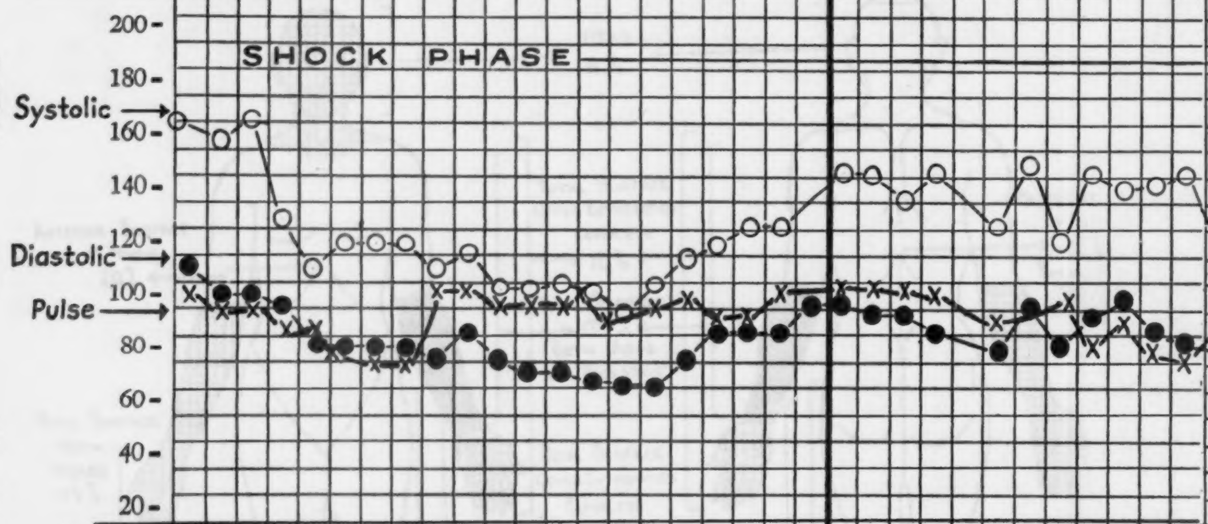
The immediate grafting of burns, as suggested by Gillies during his recent tour, is interesting, but practical only for the small, deep and definite burns. Then, as Gillies says, the burn can be "cut away" and the resulting surgical wound grafted. It is impractical in larger burns, where shock and toxic phases would prohibit such surgery.

One might summarize the salient points and reasons for the early grafting of suspected or definite deep skin burns as follows: (1) The surface is

Time of injury 8.30 A.M. Age 70 Ward L Service DR. GURD.

DATE MAY 29/42 OPERATION MECHANICAL CLEANING ANAESTHETIC NONE

TIME MAY 29 Min., Hr., Day: 10 15, 10 25, 10 45, 12 35, 3 P.M., 4, 5, 6, 7, 8, 9, 10, 11, 12, 1 AM, 2 MAY 30TH, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 1 P.M., 2, 3, 4, 5, 6, 7



R. B. C.	
W. B. C.	
Hb. %	102% 103% 112% 108% 112% 115% 105% 115% 110%
Spec. Grav. Bld.	1.0205
Hematocrit	50%
Serum Protein GM%	4.63 5.39 A./G. 3.64/1.12
Temp.	98⅘ 99⅘ 99⅘ 99⅘ 99⅘ 98° 99°
Resp.	20 18 18 18 22 18 18
Blood	
Serum or Plasma, Etc.	500 CC. + ✓ 600 = = 1100 ✓ → 500 CC. ✓ + 500 CC.
Glucose or Saline	700 CC.
Certin	
Chemotherapy	S.T.E. DRESSINGS TO FACE – MASK
Other (Local)	DRESSINGS TO HAND c̄: PLASTER MOULD
Fluids Orally	1900 CC. WATER & ORANGE JUICE c̄ GLUCOSE ←
Total Intake	
Urine Vol.	300 CC. 1300 CC.
Vomiting	NIL
Cyanosis	?
Pallor	YES
Sweating	YES
Thirst	YES
Skin Temp.	
Mental State	EXCELLENT
Restlessness	NONE
Distention	NONE
Urinalysis	NEG.

CHART 1.

SHOCK OR BURN RECORD

RD. 8.30 A.M. Age 70 Ward L Service DR. GURD.

)/42 OPERATION MAY 31/42 ANAESTHETIC JUNE 1/42

3 4 5 6 7 8 PM 9 10 11 12 1 AM 2 3 4 5 6 7 8 9 11 12 1 P.M. 2 3 4 5 6 7 8 9 10 11 12 1 A.M. 2 3 4 5

→ TOXIC PHASE

FEVER ONSET

FEBRILE

110% 100% 90% 88% 90% 86% 80% 80% 76% 73%

98° 98° 99° 99° 100° 101° 101.3° 101°

18 18 20 22 22 22 22 20

Inc. 500 cc. 500 cc.

100 cc. 1000 cc.

900 cc. 1750 cc.

6200 cc. 3250 cc.

1300 cc. 1300 500° + 1050

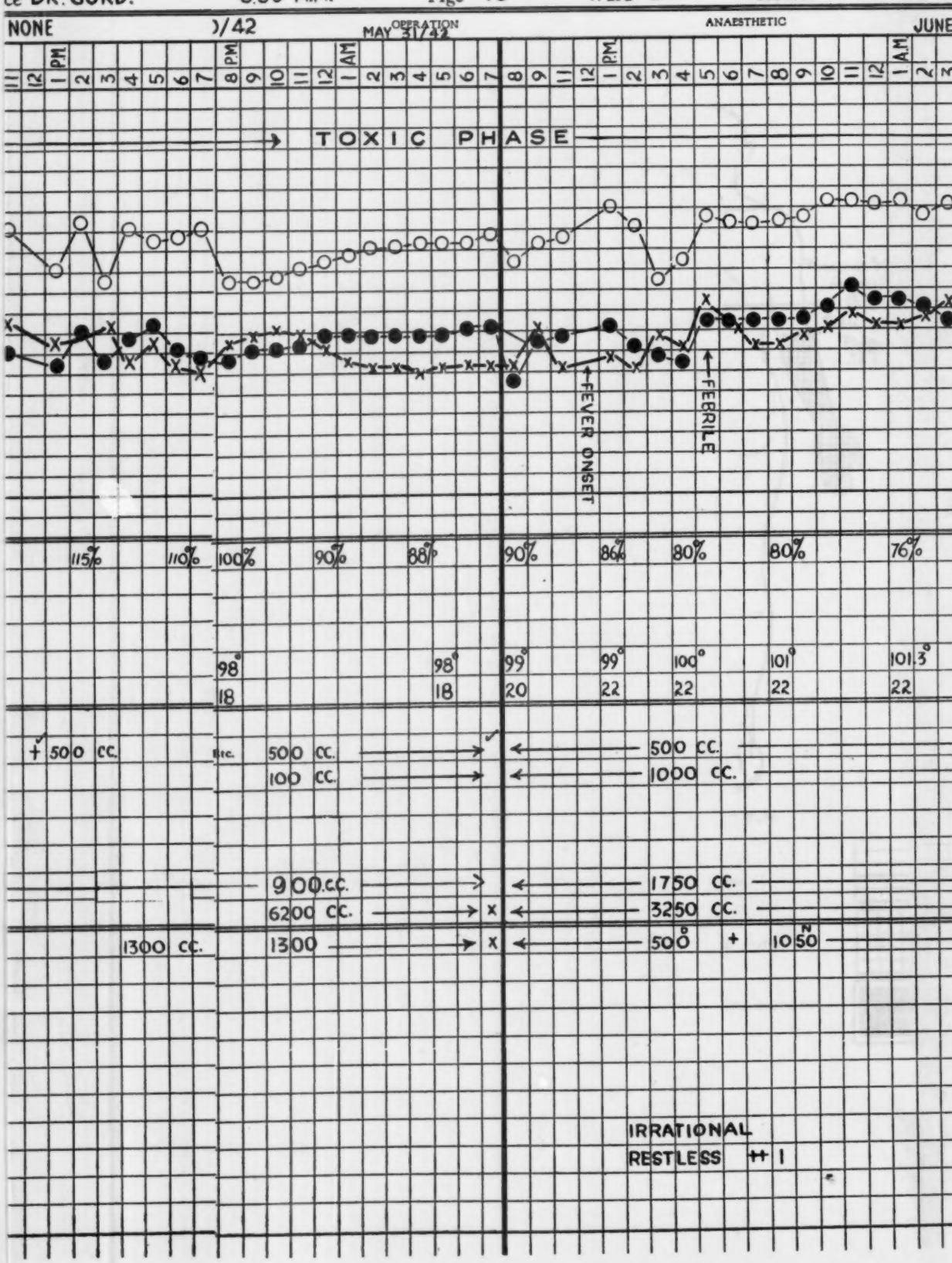
IRRATIONAL

RESTLESS ++ I

CHART 1.

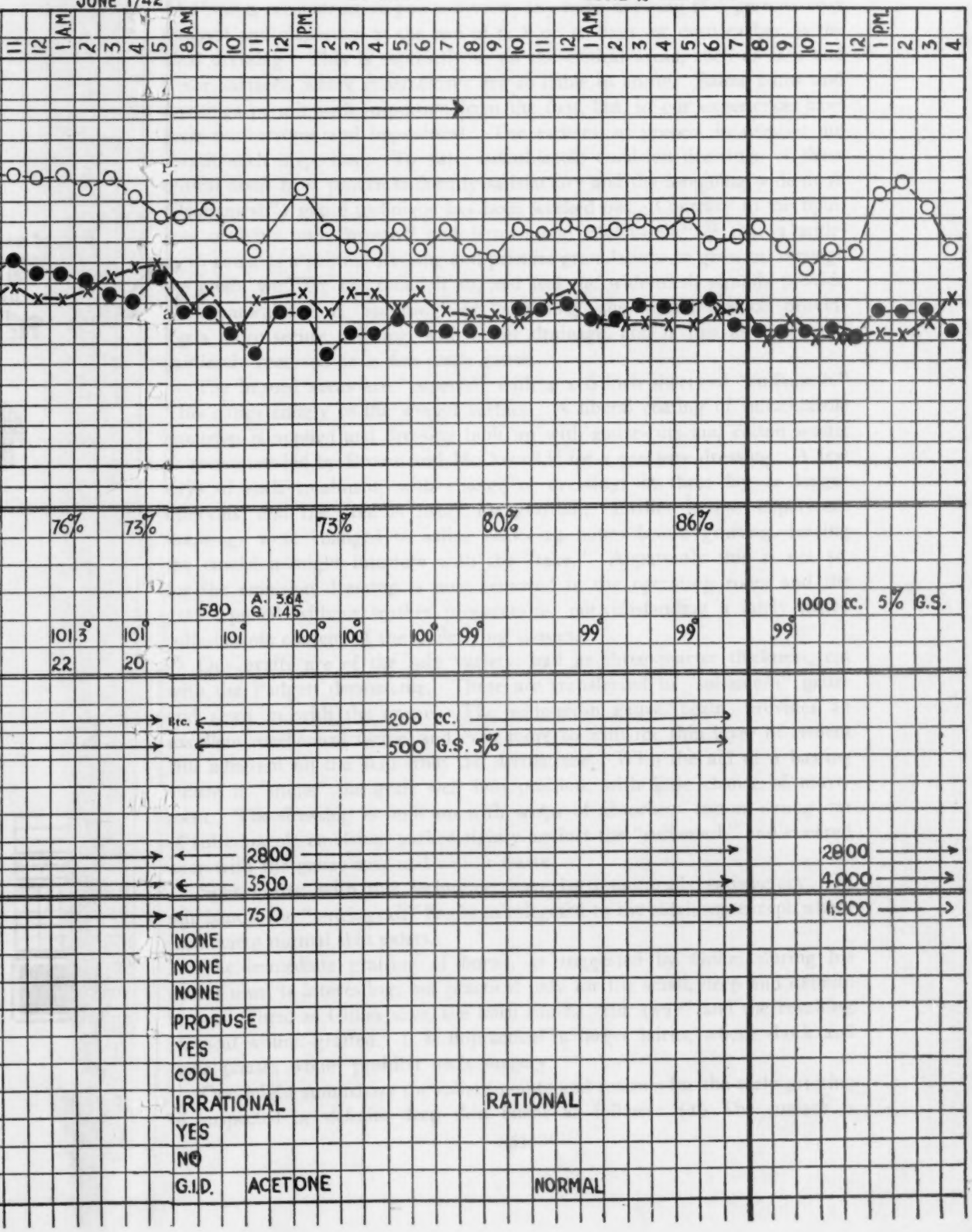
SHOCK OR BURN RECORD

ce DR. GURD. 8.30 A.M. Age 70 Ward L Service DR. GURD.



DR. GURD. 8.30 A.M. Age 70 Ward L Service DR. GURD

JUNE 1/42 OPERATION JUNE 2 ANAESTHETIC



fresh and firm, with thin granulations or beginning granulations, which will result in a minimal layer of scar tissue in the subgraft area. (2) These early surfaces are in an optimum phase for grafting As the granulations age, they tend to become exuberant and less certain for graft "take" and may have to be shaved away. (3) The grafting is clinically beneficial to the patient, as dressings and open surfaces are early done away with. (4) Infection is controlled early with sulfathiazole emulsion. (5) Any slough can be removed surgically just prior to putting on the graft. (6) It does no harm to cover up small "probable" islands of regenerating epidermis. They will at best provide a poor epithelium, at a slow rate. (7) If the patient is left with granulating areas for a long time he tends to become anemic and a less suitable recipient for his own grafts. (8) At about the fourteenth day the patient's general condition is usually at its optimum phase.

Regarding those cases in which tannic acid–silver nitrate eschar has been used, certain comments are necessary: All blebs require removal and treatment with the emulsion. One must be constantly on guard to recognize infection under the tan, and to provide adequate drainage. After the fourteenth day removal of the tannic, or any other, eschar is desirable, for early grafting of resultant raw surfaces. Usually a tannic acid eschar is beginning to separate at such time. In this connection, our experience with gentian violet or triple dye eschars has been that they seem to have become organized with the underlying tissues, and having penetrated them, are removable only through suppuration or surgery.

OBSERVATIONS ON THE PATHOLOGY OF BURNS

It is not our intention to enter into a full discussion of the pathology of burns. For this, reference may be made to the excellent papers by Brown and McDowell,[12] and by Wilson, MacGregor and Stewart.[36] We do wish, however, to make some general remarks upon the degree and healing of burns and to illustrate certain points in relation to skin grafting in cases treated with sulfathiozole emulsion.

Skin is a highly specialized structure composed of the epidermis and its appendages, and the tough fibro-elastic derma well vascularized and innervated. It is this tough elastic derma that forms the admirable bearing pad and a suitable underlay for the proper development of the epidermis.

In the local treatment of burns it is highly desirable to have the defect reconstituted, as nearly as possible by normal skin, in order to give a good functional surface and to prevent deformities.

The failure to attain this result is due to one thing, namely, the organization of granulation tissue into scar tissue. It is scar tissue that produces contractures. It is the epidermization of scar tissue that produces the poor-bearing surface of hyperkeratotic "scar skin" (Fig. 6) that binds, shortens, cracks, peels under the every day trauma of ordinary activity, and is useless as a bearing surface for labor. It is stiff and devoid of elastic tissue, is poorly vascularized, and poorly innervated. The epidermal appendages are

9 was taken.
Fig. 11.—Deep skin burn at 50 days—treated with sulphathiazole emulsion.

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FIG. 6. FIG. 7. FIG. 8.

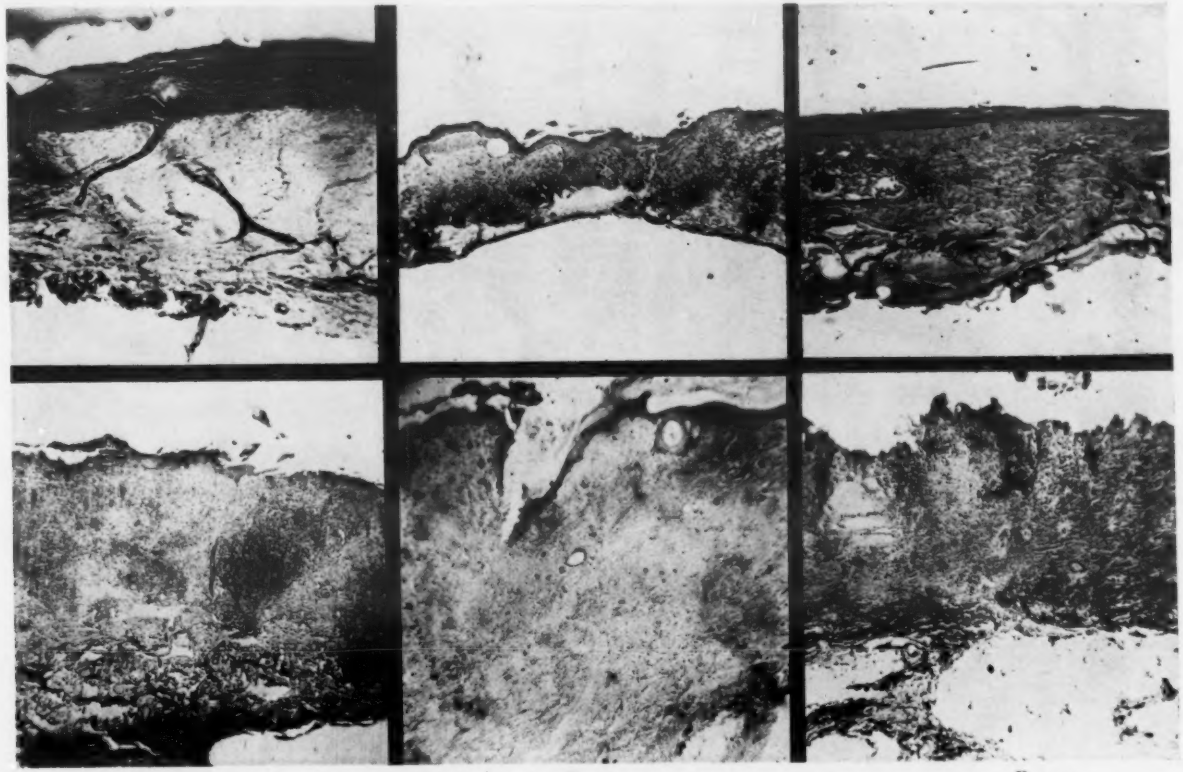

FIG. 9. FIG. 10. FIG. 11.

FIG. 6.—"Scar skin" in deep skin burn on the dorsum of the hand treated by tannic acid. Biopsy taken eight months after the accident, while the scar skin was being removed in preparation for skin grafting. At the base of the section there is a thin layer of preexisting skin with sweat glands. This is overlaid by a dense thick scar which is covered by a uniform layer of epidermis, without papillae. On the surface of the epidermis there is a very thick layer of keratin. Note the well-developed sweat gland duct extending from the epidermis through the scar toward the nest of sweat glands at the base of the section.

FIG. 7.—Regeneration of epidermis from a hair follicle in a superficial burn treated with sulphathiazole emulsion. Biopsy taken on seventh day. Note the epidermis spreading out from the broad follicle in the center of the section.

FIG. 8.—Four-day-old infected burn. Biopsy taken on ninth day, after five days' treatment with sulphathiazole emulsion.

FIG. 9.—Deep skin burn at 26 days—treated with sulphathiazole emulsion.

FIG. 10.—Junction of ungrafted area 60 days old. Skin graft 34 days old on the site from which the biopsy shown in Figure 9 was taken.

FIG. 11.—Deep skin burn at 50 days—treated with sulphathiazole emulsion.

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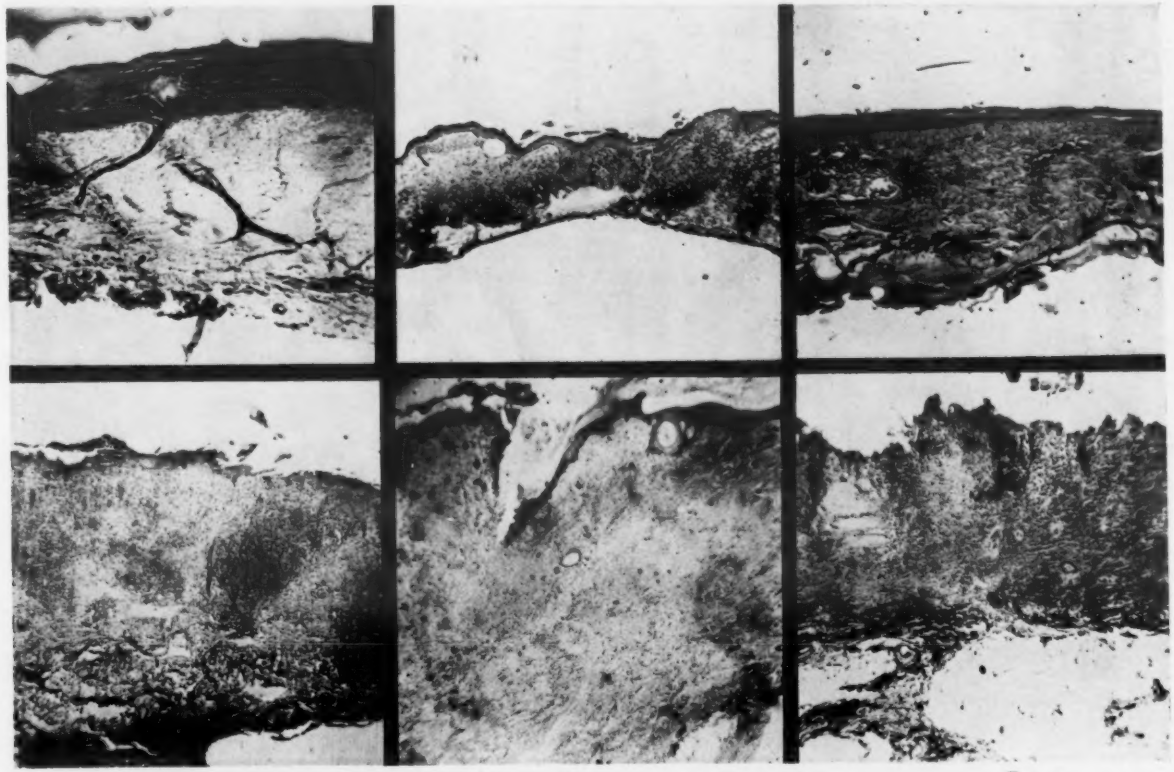

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FIG. 11.—Deep skin burn at 50 days—treated with sulphathiazole emulsion.

few or absent. The presence of too much scar tissue underlying a skin graft robs it of much of its good functional result.

Regeneration of the epidermis of burned skin takes place from viable epithelium in the area, i.e., from the margins of the wound, from hair follicles (Fig. 7), ducts of sweat glands (Fig. 9), and epidermis that has escaped complete destruction in the burned area. It is obvious that if the whole thickness of the skin is destroyed the only possible source of restitution of the epidermis is from the margins of the wound.

Healing by granulation tissue usually goes on to scar tissue formation, but in partial damage of the derma if epithelization is rapid enough, the granulation tissue appears to resolve and is replaced by reconstituted elastic derma.

The healing of burns, then, resolves itself into a race between granulation tissue formation and the regeneration of the epidermis. Upon the outcome of this race will depend the necessity or not of grafting.

Infection inhibits epithelization, and further destroys the skin and favors granulation tissue, so that the control of infection is of great importance.

Burns, from the point of view of healing, fall into two classes: (1) Those that reestablish good skin and do not require grafting. (2) Those that fail to heal, or heal by scar skin and will require grafting.

In these classes there are two extremes that are at once obvious: First, the very mild burn, in which the epidermis is not completely destroyed. Restitution is rapid. Second, very deep burns, which obviously extend beneath the derma. Healing is always by epithelization of scar tissue, and if the wound is large enough, grafting will be necessary.

Between these two extremes lie the problem cases, in which it is often difficult, on first observation, to determine the depth of the burn. Very often the depth of destruction is not uniform, so that it is not easy to predict how many possible sources of epithelial regeneration remain, or just what reaction the wound will follow. Time is required to settle this question. Usually, it can be pretty well determined at the time of the first dressing at the end of one week.

In those cases in which only the epidermis has been destroyed, or where the derma is involved only superficially, epithelization will be found complete, or nearly so, at the time of first dressing is done. There is usually some persisting inflammatory cellular exudate about the vessels.

Figure 8 represents a four-day-old infected burn, which has extended into the papillary zone of the derma. The biopsy was taken on the ninth day, after five days' treatment with sulphathiazole emulsion. Epithelization was almost complete. Note the tapering layer of epidermis which extends from a hair follicle just out of the field to the left. The derma is intact, with no granulation tissue on the surface. The epithelium is being applied directly upon the elastic derma. There is an overlying thin layer of coagulated exudate which has slightly separated. There is no edema, and only a mild perivascular cellular exudate of lymphocytes and eosinophils.

Where the burn has extended deeper, destroying the more superficial hair follicles and perhaps some of the sweat glands, the inflammatory reaction is much more intense, granulation tissue is more abundant; epithelial regeneration is slower in making an appearance; and the foci of new epidermis will be fewer in number.

Figure 9, taken on the twentieth day from the case reported herein, illustrates this degree of burn. At the base of the section there is intact viable derma containing many sweat glands. This is overlaid by a layer of granulation tissue containing areas of intense inflammatory cell infiltration, fragmented elastica, some foreign body giant cells about débris of epithelium, and of hairs left naked by destruction of the epithelial cells of the follicle. Lying perpendicularly, about the centre of the section, can be seen a regenerated sweat gland duct which, in the many sections taken, could be traced to the surface, where there is a small island of regenerating epidermis. On the surface there are ragged fragments of coagulated exudate. In other fields of this section, somewhat larger islands of epidermis were seen extending out from viable follicles.

Skin grafts were applied at this stage to part of this wound, as it was apparent that epidermization would be a very prolonged process, and scar tissue would form long before it could be completed, resulting in "scar skin" as in Figure 6.

At 40 days, another area was grafted, and at 66 days, when the remainder of the area was to be grafted, a biopsy was taken through the first graft, 34 days after its application, and the adjoining ungrafted area. Figure 10 shows the junction of the ungrafted area and the first graft. There is a marked increase in the granulation tissue in the ungrafted area. The grafted area shows a remarkable transformation. The graft and the skin layer of original derma are joined by a narrow zone of young fibrous tissue, in which there are a few foci of lymphocytes and some foreign body giant cells about groups of epithelial cells. Most striking is the effect upon the damaged granulating skin by the application of the graft. No paring was done.

Figure 11 represents a section from another burned area in the same man as in Figures 9 and 10, at 50 days. The section, stained with Weigert's elastic tissue technique, shows subcutaneous tissue, and a very narrow strip of derma, represented by the dark-staining elastica at the base. There are no viable sweat glands or hair follicles and no evidence of epithelial regeneration in this section. Overlying the thin layer of derma is a thick, irregular layer of granulation tissue showing marked inflammatory cell infiltration and areas of edema. Although the derma is not completely destroyed one could not expect much of epithelial regeneration. Skin grafting was done at this stage after paring down this type of excessive edematous granulation tissue, which is not a satisfactory bed for the reception of a graft.

ILLUSTRATIVE CASE HISTORY

Case Report.—M. G. H.: O. G., male, age 70, was admitted, May 28, 1942, about two hours after a severe flash burn of the face, neck, both forearms and hands. An accurate estimate of the area and depth of burn will be found on the accompanying burn chart. In this connection, it should be noted that two weeks elapsed before it could be definitely decided that the left forearm required skin grafting.

On admission, the patient was in moderate shock, clinically, and appropriate measures were instituted. Local treatment was carried out with sulfathiazole emulsion and pressure dressings, according to our timing chart. The details of this and the details of his general course during the first four days will be found on the burn chart in some detail. Certain points about this period, as noted on the chart, deserve special comment. In the first place, the shock was controlled by plasma, *etc.*, though at the 10- to 18-hour period, with some difficulty. This was occasioned by the difficulty with the continuous drip, which infiltrated, and through a misunderstanding was discontinued for three hours. On rectifying this, when the hemoconcentration was reported, no further trouble occurred during the shock period. This serves to illustrate forcibly, and we have unfortunately had other such confirmation, the necessity of maintaining a 24-hour service by the shock team. At 45 hours, after apparently being well controlled, a definite toxemia phase developed quite suddenly, with fever, rapid pulse, restlessness, irrational mental state, partial suppression of urine and rapid fall in hemoglobin. With the administration of intravenous glucose this condition rectified itself in about 15 hours. Following this period, there will be noted a rapid return to relative normality in all recordings and in his general condition.

The patient's subsequent course was uneventful and without any infection, although the depth of the burn on his left forearm was only determined after two weeks. Even at this time it was determined with some difficulty, since many epithelial islands were evident. A further delay of 10 days ensued, as Doctor Gerrie was out of town. On the 26th day split-grafts were placed *in situ* without any special preparations, and 100% "take" ensued. There is no functional disability.

During the skin-grafting period biopsy studies were made before and after grafting, the interesting results of which are referred to in the section on pathology.

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A STUDY OF THE "SHOCK-DELAYING" ACTION OF THE BARBITURATES

WITH A CONSIDERATION OF THE FAILURE OF OXYGEN-RICH ATMOSPHERES TO DELAY THE ONSET OF EXPERIMENTAL SHOCK DURING ANESTHESIA

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IT HAS BEEN SHOWN REPEATEDLY that there is a concentration of the cellular elements of the blood under ether and a dispersion of them under various barbiturates; the blood has thus been described as concentrated under ether and "diluted" under barbiturates. Because of these and other observations, Seeley, Essex and Mann[15] (1936) set out to determine by experiment whether the onset of traumatic shock would occur after the same interval under ether as under a barbiturate. A first step in their work was to develop a "standard method" of producing trauma.

We have repeated this work, insofar as their two major groups of dogs are concerned. The possibility that barbiturates will delay the onset of shock is of great importance if it is applicable to the shock problem in general, and of considerable importance if this effect can be shown to hold even under special circumstances. We have examined the effect of a barbiturate and compared it with ether in circumstances where shock is produced by bleeding. We have also studied the effects on "shock-time" of respiration of room air compared with oxygen-rich atmospheres under the conditions of these experiments.

METHODS.—*Experimental Animals:* Forty mongrel dogs were used in this study.

Anesthesia.—A freshly prepared 5% solution of sodium amytal was injected into the saphenous vein in an initial dose of 50 mg. per Kg. Ether was induced in a closed cabinet and then maintained as described by Hardenbergh and Mann[7] (1927) in order to repeat the experiments of Seeley, Essex and Mann. Particular effort was made to maintain comparable levels of anesthesia under the two anesthetics. The level chosen was that in which the corneal reflex persisted but was very sluggish. In about half of the experiments of each group the level of anesthesia was controlled further by means of comparable records of the flexion reflex of the leg when the central end of the cut sciatic nerve was stimulated by a Grass stimulator (condenser type); see Beecher and Moyer,[3] 1941.

The Production of Shock.—The intestinal manipulation experiments were carried out as described by Seeley, Essex and Mann (1936). They produced trauma in the following way:* "The entire length of small intestine was delivered outside the abdominal cavity and gently manipulated by a continuous rolling motion between the hands of the operator. After 30 minutes of manipulation the intestines were spread out on towels on the anterior abdominal wall. The intestines were turned every 30 minutes to remove fibrin and to avoid unequal exposure of the loops. The blood pressure in the femoral artery was recorded at intervals on a standard kymograph. When the blood pressure had declined to a level of 70 Mm. of mercury, the animals were considered to be in a state of shock." Blood concentration studies were made.

In the bleeding experiments which we carried out, the first blood was drawn from the femoral artery as soon as the control samples and measurements had been taken. The first hemorrhage amounted to 1.0% of the body-weight. Thirty minutes later a second hemorrhage of 0.5% body-weight was carried out. Every half-hour following this, until death, blood to the extent of 0.25% body-weight was withdrawn.

"Shock-time" was measured from the beginning of intestinal manipulation, or in the bleeding experiments from the beginning of bleeding, until the mean arterial blood pressure had fallen to 70 Mm. Hg. and had remained at this level or below for one-half hour. "Death-time" was measured from the beginning of intestinal manipulation, or from the beginning of bleeding in the bleeding experiments, until death occurred.

Blood Data.—Mean arterial blood pressure was determined and recorded through a cannula placed in the femoral artery. Hematocrit determinations in the bleeding experiments were made by the method of Sanford and Magath[13] (1929) on 6 cc. arterial samples. Blood withdrawn routinely in the course of the experiment was utilized for this determination. In the intestinal manipulation experiments the hematocrit was determined on 1.25 cc. venous blood by the method of Rourke and Ernstene[12] (1930). Serum protein measurements were made with the Zeiss refractometer. This instrument had been repeatedly checked against Kjeldahl protein determinations. Arterial and venous blood oxygen measurements were made by the method of Van Slyke and Neill[19] (1924). When ether was present in the blood the modifications of Shaw and Downing[16] (1935), and Snyder[17] (1938) were used in determining oxygen content of the blood.

Results.—The data of Table I have been compiled from the results of Seeley, Essex and Mann. Tables II and III present our results on repetition of the same experiment. It can be seen that in the case of the ether shock time we are in remarkably close agreement: Our data differ from theirs by

* Parsons and Phemister[10] (1930) used exposure and repeated manipulation of the intestine to produce shock, apparently under barbiturates and ether, but they record few details of their study.

only 5%.* It is worth observing that in this case both groups had the same number of experiments, seven. In the case of the sodium amytal (alone) experiments we had the same number of experiments as with ether, seven; while Seeley, Essex and Mann had three. Possibly this may account in part at least for our failure to agree better with their amytal data. A further explanation probably lies in depth of anesthesia. Seeley, Essex and Mann gave, apparently, a single initial injection of the barbiturate without later supplement.†

TABLE I

DATA COMPILED FROM ARTICLE BY SEELEY, ESSEX AND MANN[15]

Anesthetic Agent	Number of Dogs	Time for Shock to Develop Hrs.	Death-Time Hrs.
Ether alone	7	Average 3.88	Average 5.88
Sod. amytal, alone, 50 mg./Kg.	3	Average 11.55	Average 14.73
Sod. amytal 25 mg./Kg., preceding ether	5	Average 10.50	Average 13.50
Ether followed by 25 mg./Kg. sod. amytal	2	One animal 6.42	One animal 10.25
		One animal 11.42	One animal 15.08

TABLE II

SHOCK BY INTESTINAL MANIPULATION

Ether and Room Air

No.	Wt. Kg.	Sex	Shock-Time Hrs.	Death-Time Hrs.	Hematocrit Control %	Hematocrit Shock %	Serum Protein Control %	Serum Protein Shock %
1.	7.2	♀	10.25	14.17	50.0	69.4	6.94	6.34
2.	7.9	♀	1.00	4.83	56.6	67.6	6.90	7.13
3.	11.0	♂	8.16	10.33	56.3	66.1	7.93	7.61
4.	8.8	♀	4.91	5.55	57.7	70.4	7.70	6.90
5.	12.6	♂	2.28	8.58	50.0	60.4	6.68	6.75
6.	12.2	♂	1.00	5.16	47.0	69.0	6.25	7.70
7.	8.8	♂	1.00	1.50	55.1	80.8	7.11	7.91
Average			4.09	7.16	53.2	69.1	7.07	7.19
			4.1 ± 1.5	7.2 ± 1.6				

*This should dispose of any possible objections that differences between the average room temperatures, mean barometric pressures, or mean relative humidities would, in altering evaporation rates, make it impossible to compare data obtained in these different parts of the country. It is our policy to observe and record such data twice daily, in the middle of the morning and in the middle of the afternoon. These factors are too often neglected. A further control on this point is the fact that both types of experiments, ether and barbiturate, were made in each place; so differences between the two should emerge, if present.

† A recent letter from Dr. Mann supplied the following information: "While in our original article we presented the data on only three dogs in which amytal was used, before the paper was published we had enlarged the number and to date have a fairly large series. We failed to state in the article that occasionally it was necessary to give small amounts of amytal when the animal became light, until shock had developed. It is also our practice to decrease ether as soon as the animal is definitely in a condition of shock (suggested by Meltzer) so that the time of action of the two anesthetic agents does not differ greatly."

TABLE III

SHOCK BY INTESTINAL MANIPULATION

Sodium Amytal and Room Air

No.	Wt. Kg.	Sex	Shock-Time Hrs.	Death-Time Hrs.	Hematocrit Control %	Hematocrit Shock %	Serum Protein Control %	Serum Protein Shock %	Na Amytal mg./Kg. Initial	Na Amytal mg./Kg. Suppl.	No. Inject.
8.	9.8	♀	11.41	14.83	32.3	57.2	7.13	8.33	51	5.1	2
9.	18.6	♂	9.00	15.58	39.6	62.6	7.13	7.72	51	24.8	18
10.	6.8	♂	9.16	10.30	40.4	52.0	6.20	6.18	50	14.7	5
11.	8.5	♂	2.17	5.83	37.6	46.5	6.66	6.10	50	19.4	5
12.	11.0	♂	9.00	18.50	39.8	60.2	7.89	8.18	50	30.0	16
13.	8.4	♂	10.50	11.16	40.4	74.8	7.09	7.70	50	11.3	4
14.	28.4	♂	6.58	8.90	53.8	67.3	—	—	50	30.0	6
Average.....			8.26	12.16	40.6	60.1	7.02	7.37	50	19.3	8
			8.3 ± 1.2	12.2 ± 1.7							

In our experience, it was not possible to maintain a level of barbiturate anesthesia that was comparable with that of the ether experiments by this single injection (except in one out of 20 amytal experiments). In our experiments, following this initial injection, sometimes sooner than an hour, the animal's anesthesia would become very light, so that the dogs could not truly be considered as anesthetized unless supplementary doses of barbiturates were given. It seems to us that Seeley, Essex and Mann were comparing ether anesthesia with, in the latter part of their barbiturate experiments, a state in which hardly any anesthesia produced by the drug itself was present, but what depression there was was chiefly the depression of the shock state. We have made particular efforts to maintain comparable levels of anesthesia. Because of the larger number of our amytal experiments and the particular efforts we have made to maintain comparable levels of anesthesia in the two groups we are inclined to believe that our values more nearly express, than does the other series, the shock-delaying properties of the barbiturates: Shock was delayed twice as long under barbiturates as under ether (our data) rather than three times as long (Seeley, Essex and Mann). There is no point in dwelling on this quantitative difference, of much more importance is the fact that we confirm their observation that the barbiturates *under the conditions of this experiment* do delay the onset of shock, and that death will occur later if barbiturate anesthesia be used than if ether is the agent employed.

We next set out to test whether or not barbiturates would delay the onset of shock produced by bleeding, with the belief that this might help to round out the picture of possible usefulness of the agents in seriously wounded individuals. From Tables IV, V, VI and VII it is apparent that no significant shock-delaying action has been demonstrated when hemorrhage constitutes the trauma.

We had planned to compare the effects of barbiturates and ether in muscle trauma experiments but decided not to, in view of the failure of barbiturates to delay shock produced by bleeding in our experiments and the failure of barbiturates to delay, in comparison with ether, the shock produced in the muscle trauma experiments of Parsons and Phemister,[10] and of Bla-

lock.[4] Parsons and Phemister (1930), in a large series of experiments (70) upon anesthetized dogs, studied the effects of stimulation of the nerves of the limb, of traumatization of the limb, and of bleeding. For anesthetics they employed ether and morphine in 24 cases, ether alone in 17 cases, morphine and barbital in 18 cases, and barbital alone in 11 cases. They report that except where morphine was used, the effects under ether or barbiturate were very similar.

TABLE IV
SHOCK BY HEMORRHAGE
Ether with Room Air

No.	Wt. Kg.	Sex	Shock-Time, Hrs.	Death-Time, Hrs.	Hematocrit Control %	Hematocrit Shock %	Serum Protein Control Gm. %	Serum Protein Shock Gm. %	Arterial O_2 Content Control Vols. %	Arterial O_2 Content Shock Vols. %	Venous O_2 Content Control Vols. %	Venous O_2 Content Shock Vols. %
1.	6.4	♂	4.50	4.85	42.1	30.5	7.96	6.19	13.7	10.4	6.0	2.9
2.	9.7	♀	2.05	4.32	51.9	39.8	6.64	5.18	19.9	16.8	6.2	4.7
3.	8.0	♀	4.00	5.09	51.8	36.8	6.55	5.45	18.1	13.4	10.8	1.8
4.	8.2	♀	1.50	2.00	49.4	41.5	6.77	5.56	18.9	14.0	8.8	2.1
5.	6.0	♀	1.63	2.67	58.9	51.5	7.22	5.94	19.0	18.5	12.0	8.6
6.	7.0	♀	1.67	4.57	46.9	43.4	6.89	5.90	18.1	16.7	16.8	9.1
	Average.....		2.56	3.92	50.2	40.6	7.01	5.70	18.0	15.0	10.1	4.9
			2.6± 0.6	3.9± 0.5								

TABLE V
SHOCK BY HEMORRHAGE
Ether with 100% Oxygen

No.	Wt. Kg.	Sex	Shock-Time, Hrs.	Death-Time, Hrs.	Hematocrit Control %	Hematocrit Shock %	Serum Protein Control Gm. %	Serum Protein Shock Gm. %	Arterial O_2 Content Control Vols. %	Arterial O_2 Content Shock Vols. %	Venous O_2 Content Control Vols. %	Venous O_2 Content Shock Vols. %
7.	11.0	♂	0.50	1.08	45.3	42.8	6.89	6.62	—	—	—	—
8.	7.3	♀	4.18	4.49	39.1	44.0	5.88	4.16	24.8	19.8	19.1	7.9
9.	7.0	♂	4.40	4.45	45.6	35.1	7.50	5.79	19.3	15.2	13.1	5.0
10.	7.8	♂	4.33	4.44	52.0	43.7	7.07	5.72	19.0	15.7	14.4	7.3
11.	9.0	♂	4.05	4.48	63.3	52.9	8.08	5.70	26.6	23.6	21.0	13.2
12.	12.5	♂	2.75	3.68	58.6	51.1	6.73	5.58	24.6	21.8	16.5	14.4
13.	9.2	♀	3.00	6.77	60.6	55.0	7.68	6.27	24.2	22.6	23.8	13.3
	Average.....		3.32	4.20	54.9	46.4	7.12	5.69	23.1	19.8	18.0	10.2
			3.3± 0.5	4.2± 0.6								
Combined ether Average.....			2.8± 0.4	4.1± 0.4								

TABLE VI
SHOCK BY HEMORRHAGE
Sodium Amytal with Room Air

No.	Wt. Kg.	Sex	Shock-Time, Hrs.	Death-Time, Hrs.	Hematocrit Control %	Hematocrit Shock %	Serum Protein Control Gm. %	Serum Protein Shock Gm. %	Na Amytal mg./Kg. Initial	Na Amytal mg./Kg. Suppl.	No. Inject.	Arterial O_2 Content Control Vols. %	Arterial O_2 Content Shock Vols. %	Venous O_2 Content Control Vols. %	Venous O_2 Content Shock Vols. %
14.	8.6	♂	1.55	—	35.6	33.8	6.98	5.97	50	0	1	14.0	14.1	10.6	6.0
15.	11.0	♂	4.90	—	29.1	28.6	5.46	4.94	50	41	7	11.0	11.1	5.9	1.6
16.	11.8	♀	4.72	—	38.4	32.9	7.27	6.12	50	13	4	11.4	13.0	7.4	2.0
17.	8.5	♀	1.55	—	39.4	39.2	4.96	4.05	50	12	3	14.2	16.0	9.6	8.2
18.	12.2	♀	0.75	1.58	38.9	54.7	6.49	6.32	50	8	2	14.9	18.8	9.1	1.6
19.	10.7	♂	5.00	7.38	42.7	53.8	6.05	5.34	56	15	5	15.8	20.1	9.0	2.2
20.	11.2	♀	5.50	6.53	43.7	42.7	5.90	4.81	54	12	9	17.7	17.3	15.5	5.6
	Average...		3.42	5.16	38.0	40.8	6.16	5.36	51	14	4	14.1	15.8	9.6	3.9
			3.4± 0.8												

TABLE VII

SHOCK BY HEMORRHAGE

Sodium Amytal with 100% *Oxygen*

No.	Wt. Kg.	Sex	Shock-Time, Hrs.	Death-Time, Hrs.	Hematocrit Control %	Hematocrit Shock %	Serum Protein Control Gm. %	Serum Protein Shock Gm. %	Na Amytal mg./Kg. Initial	Na Amytal mg./Kg. Suppl.	Na Amytal mg./Kg. No. Inject.	Arterial O_2 Content Control Vols. %	Arterial O_2 Content Shock Vols. %	Venous O_2 Content Control Vols. %	Venous O_2 Content Shock Vols. %
21.	9.0	♀	4.50	5.36	42.9	38.8	7.57	6.68	50	47	11	18.7	16.5	10.1	2.8
22.	9.0	♀	3.00	6.60	44.0	48.0	7.11	5.70	55	24	8	19.6	21.8	14.7	9.6
23.	7.0	♀	0.50	6.77	43.7	53.4	5.05	4.73	54	20	6	18.7	22.7	12.5	7.0
24.	9.7	♀	2.50	6.07	37.9	37.1	5.53	4.51	54	16	7	13.7	16.6	9.8	6.2
25.	8.2	♀	4.00	5.67	43.0	42.7	7.27	6.14	54	28	13	15.8	15.3	14.5	6.5
26.	8.0	♂	4.50	5.20	32.4	39.4	6.93	6.01	52	18	8	14.0	16.6	11.8	5.7
Average...			3.17	5.95	40.7	43.2	6.58	5.63	53	26	9	16.8	18.3	12.2	6.3
			3.2± 0.6	6.0± 0.3											
Combined Na amytal Avg.....			3.3± 0.5	5.7± 0.6	39.3	41.9	6.35	5.49	52	20	6				

Blalock (1942), in his study of the comparison of the effects of the local application of heat and cold in the prevention and treatment of shock produced by pounding an extremity, records among other data the following when heat was added to the injured member (Table VIII):

TABLE VIII

Anesthetic Agent	Nembutal	Barbital	Morphine and Ether
Number of dogs	14	6	5
Av. diff. in wt. of traumatized and nontraumatized parts, in per cent body-wt.	3.80	4.05	3.40
Death-time	5° 52′	5° 44′	5° 50′

It is clear that the barbiturates do not prevent in comparison with morphine and ether the loss of fluid into the traumatized extremity, under the conditions of this experiment, nor do they delay the death-time over that produced under morphine and ether. Therefore, considering our hemorrhage data, and Parsons and Phemister and Blalock's muscle trauma data, we have not continued with the traumatization of limb experiments.

Conflicting reports have been made concerning the usefulness of oxygen in shock. We wished to test whether or not breathing of an oxygen-rich (about 100%) atmosphere would be of value in delaying shock under conditions where these two widely different types of anesthesia were employed. From the data, it is apparent that it is possible nearly to double the venous oxygen,* and yet we do not find any significant delay in the onset of shock as a result of using an oxygen-rich atmosphere. Since this was so, we have combined the high oxygen and the room air data.

* For these experiments blood was withdrawn from the femoral vein. It seemed to us that the question of whether the oxygen content could be raised in this peripheral venous blood constituted a severer test than that provided by mixed venous blood from the right heart. We, of course, would have added such determinations had we not failed to get delay in the shock-time as a result of the high oxygen atmosphere, notwithstanding the great increase in the oxygen content of the femoral venous blood.

DISCUSSION.—While it is true that most of the methods employed for producing experimental shock involve a complicated and confusing variety of traumatic stimuli in a single procedure, it is evident that the method of Seeley, Essex and Mann has this objection. The fact that clinical shock may be due to several simultaneous causes in a given patient, is no indication for needlessly complicating experimental procedures. Admittedly, the problem of how to produce experimental shock is a most difficult one; however, it can be pointed out that the intestinal manipulation method of producing traumatic shock involves at least four types of stimuli well known to lead to or aggravate the condition of shock: Tissue trauma, harmful nerve stimulation, chilling, plasma loss and dehydration. The last factor appears to be of major importance in the shock developed by this method. The multiplicity of these factors perhaps accounts for the variability of results obtained from one experiment to another, not only in our hands, but in the experiments reported by the originators of the method (cf. the large standard errors of the mean).

We have available the data of Seeley, Essex and Mann, and the confirmatory data of Kendrick[8] (1939). These studies seem to have demonstrated that real delay in the onset of shock *under the special circumstances of this experiment* can be effected by barbiturates *in comparison with ether.* Our own data support this conclusion.

The major purpose of this communication, and the questions we wish to raise, are concerned primarily with the important assumptions that have been made by others concerning the implications of the work of Seeley, Essex and Mann. It has been assumed by numerous writers, on the basis of the report by Seeley, Essex and Mann, (although not by these men) that it is safe, and desirable, to recommend the use of barbiturates in wounded men with the aim of delaying shock, however it may be caused. Various references to bear this out could be given. More to the point in the present military situation is the official report of the Tenth International Congress of Military Medicine and Pharmacy held in Washington, D. C., May 7–15, 1939. On page 188, the statement is made that *"these experiments* (of Seeley, Essex and Mann) *indicate that patients to whom sodium amytal is administered early, more often survive the exposure, delay and transportation incident to their evacuation to installations of definitive treatment."* (Italics ours). This report goes on to say that *"with these facts in mind it seems advisable to equip battalion surgeons and collecting company personnel with sodium amytal*—(to delay) *the onset of shock . . . ."* The experiments under discussion may or may not be applicable to man subjected to the common types of shock producing trauma of the battle field.

Transference of the findings of Seeley, Essex and Mann, obtained from studies on anesthetized dogs to a general recommendation concerning the equipment of battalion surgeons and collecting company personnel for widespread application to seriously wounded soldiers, involves a good many assumptions. The interpreters of the Seeley, Essex and Mann data make at least two major suppositions that are difficult, if not impossible, to uphold:

First, to have made the recommendations mentioned, it must have been assumed by the interpreters that the findings of Seeley, Essex and Mann (which they obtained in their specialized method of producing shock) hold for battle field shock in general; at least they must have supposed that the recommended barbiturate will not of itself constitute a real hazard. Evidence directly opposing both of these opinions is at hand. Second, use of the data obtained from the anesthetized dogs as a guide for treating wounded but unanesthetized men requires the assumption that the etherized dog represents unanesthetized man and that the dog under the influence of the barbiturate is comparable to man following the administration of barbiturates. In other words, it must be recognized by those who wish to transfer these findings to man that the data of Seeley, Essex and Mann do not compare an unanesthetized group of dogs with a group of dogs under a barbiturate. The comparison is between dogs under ether and dogs under a barbiturate. It would be a more accurate transference of the data in question if those who wish to apply these data to man would conclude that the wounded soldiers would be better off on receiving barbiturate than he would be if he received ether. Unfortunately, data are not available which permit the comparison of shocking stimuli with and without anesthesia; the available data merely present comparisons between the effects of one anesthetic agent and another. It seems apparent that recommendation of the general use of barbiturates for the prevention of shock in severely wounded soldiers, by battalion surgeons and collecting company personnel, is not securely founded, and there is considerable evidence that such use of the barbiturates may be dangerous.

While barbiturates delay the onset of shock in the intestine-manipulating experiments of Seeley, Essex and Mann, of Kendrick, and in ours, it must be recognized further that this is a special type of trauma, and all subjects are under anesthesia. As pointed out by Seeley, Essex and Mann, the method is probably effective chiefly through its dehydrating effect. The barbiturates appear to be more effective in preventing water and plasma loss from the exposed intestines than is ether. The chance that the difference between the two agents may appear simply because ether may increase this loss above normal or above that which is the case under barbiturates must be kept in mind. Seeley, Essex and Mann observed, as we have also, that the loss of fluid from the surface of the traumatized intestines was much less rapid under the barbiturates than under ether.

Other evidence could be cited to support the view that the plasma volume tends to be preserved or increased under the barbiturates whereas the reverse is true under ether. (Ref. Hamlin and Gregerson[6] (1939), McAllister[9] (1938); Searles and Essex[14] (1936), Adolph and Gerbasi[1] (1933), Bourne, Bruger and Dreyer[5] (1930), Barbour and Bourne[2] (1923) and others). Polderman and Beecher[11] (1942) have shown that the volume flow of cervical lymph is usually about 70 per cent greater under ether than under barbiturates. This adds one step to the probable explanation of the greater loss of fluid under ether and the more rapid decline in the

subject's condition under this agent than when under the barbiturates, under the special circumstances of this experiment.

It must be emphasized, that if the barbiturates are effective only by virtue of their antidehydrating effects, then one could hardly expect them to be of value in shock due to other causes, as hemorrhage, tissue damage and so on, unless rapid fluid loss from large surfaces was a complicating factor. In these other types of shock it is reasonable to suppose that the barbiturates might be distinctly harmful, for their typical effects are undesirable—depression of the respiratory volume, decrease in, even loss of effectiveness of the normal respiratory stimulant, carbon dioxide (ref. Beecher and Moyer[3] (1941) for a discussion of this and references to other papers on the subject), depression of blood pressure, depression of the cardiac muscle, *etc.* These and many other effects of the barbiturates could be listed to emphasize that unless specifically indicated these agents had better be avoided in the seriously wounded.

SUMMARY AND CONCLUSIONS

1. We have confirmed the observation of Seeley, Essex and Mann that shock produced in dogs by exposure and manipulation of the intestines is slower to appear when barbiturate (sodium amytal) anesthesia is used than when ether anesthesia is employed.

2. No significant delay was found in the onset of shock produced by hemorrhage when barbiturate (sodium amytal) anesthesia was compared with ether anesthesia. This observation coupled with those of Parsons and Phemister, and Blalock, who found similar effects under barbiturate and ether anesthesia when shock was produced by muscle trauma, indicates that the barbiturates as compared with ether anesthesia are not useful in delaying all types of shock.

3. On the basis of the available evidence, the barbiturates appear to delay shock in comparison with ether, only when the chief shocking trauma is dehydration or plasma loss from wound surfaces.

4. Numerous recommendations have been made, on the basis of the work of Seeley, Essex and Mann (but not by these men), that barbiturates be administered to all wounded men if the development of shock is anticipated. Such recommendations involve two major assumptions, both of which, on the basis of the available information, are untenable: First, the numerous recommendations that barbiturates be administered routinely to all seriously wounded men involves the assumption that the barbiturates will be of value in shock, *however caused*; if one can judge by the results in dogs, this is not the case (see above); or at least the assumption is made that such administration of barbiturates will not be dangerous. Abundant evidence is available to indicate that this is not the case. Second, direct application as a shock preventive, of the observation mentioned in paragraph 1, above, requires the assumption that comparison of the barbiturate data with the ether data is the same thing as a comparison of barbiturate data with a condition of no anesthesia, certainly not the case. No data are available to indicate that the onset

of shock is slower under barbiturates than in unanesthetized subjects.

5. In the experiments presented here, the administration of approximately 100% oxygen did not significantly delay, in comparison with room air, the onset of shock due to bleeding under either a barbiturate or ether, notwithstanding great elevation in peripheral venous blood oxygen content as a result of breathing the high oxygen atmosphere.

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CHEST INJURIES*

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THE FOLLOWING was originally assembled to serve as a supplement to our colored moving picture "Chest Injuries." These films are to be used in connection with the instruction of Army medical officers. However, in war time it is important for *all* physicians to be familiar not only with the clinical diagnosis of the various chest injuries, but also with the underlying pathologic physiology of the heart and lungs produced by these injuries. With this knowledge the therapy will then seem simple and logical. The immediate first-aid treatment to be administered at the site of the injury is presented first, followed by the indications for, and technic of, the more specialized hospital procedures. Finally, we state the indications for surgery, the immediately corrective as well as those for the late sequelae. Thus, in carrying the management of the patient from the place of injury through the specialized surgery that chest injuries entail, a practical compendium of thoracic trauma is presented. Such an outline should be helpful to all physicians who are called upon to handle chest injuries as well as those who may eventually study the film.

This material is presented in outline form, with the minimal amount of explanation possible. The discussion is confined exclusively to the special therapeutic problems that arise in the management of chest wounds. The treatment of shock and general care of the injured are not included.

GENERAL PRINCIPLES

Impairment in the functions of the thoracic organs produces profound changes in the general physiology of the body. Therefore, chest injuries demand immediate treatment, which differs widely from that employed in other regions. At the outset, the problems are primarily those of mechanical derangement of the thoracic organs and hemorrhage, while later infection assumes a dominant rôle. Certain general principles must be kept in mind in treating chest injuries.

It is necessary:

(1) To remove, widely and very gently, the clothing over the chest, so that exposure adequate for thorough examination may be obtained.

(2) To promptly restore the normal relationship of the intrathoracic organs, which of themselves, may be largely responsible for shock.

a. Cover open chest wounds.

b. Control pneumothorax.

c. Control cardiac tamponade.

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(3) To give immediate attention to shock and hemorrhage.

(4) To avoid movement of the injured until he can be transported to a place where hospital care may be administered.

(5) To watch constantly for the further accumulation of air or fluid in the pleural or pericardial cavities.

(6) To preserve the cough reflex by judicious sedation.

(7) To keep the bronchial tree free of blood and secretions.

(8) To support the chest wall where there is paradoxical or painful movement.

(9) To prevent or minimize sepsis—the direct or indirect cause of practically all late morbidity and mortality.

(10) To establish at once a reasonably high blood sulfonamide level, preferably sulfadiazine (8 to 10 mg. per cent), by the administration of the sodium salt intravenously.

(11) To give precedence to the treatment of the chest wound in cases of multiple injuries of various parts of the body. Only the control of hemorrhage precedes the attention to the thoracic injury. However, in combined thoraco-abdominal wounds, every attempt must be made to improve the thoracic physiology so that abdominal exploration can be undertaken promptly.

(12) To employ, invariably, positive pressure anesthesia when open operations of the thorax are indicated.

INJURIES REQUIRING CONSERVATIVE CARE

I. Thoracic Concussion (*Commotio Thoracis*):

Cause: Nonpenetrating chest injury (exploding shells) produces a condition similar to cerebral concussion. Autopsies show no cause of death. Emotional strain and vasomotor reflexes are considered factors.

Signs and Symptoms: Cold, clammy skin; slow, thready, irregular pulse; sighing respirations; unconsciousness.

Treatment: As for shock. (Oxygen of some value)

Prognosis: If patients survive for a few minutes, they usually recover.

II. Traumatic Asphyxia:

Cause: Sudden, violent thoracic compression producing wave of back pressure, by way of the superior vena cava, dilating peripheral veins. This is followed by venous stasis and loss of vascular tone. Petechial hemorrhages frequent.

Signs and Symptoms: Deep violet-blue discoloration of face and neck with edema, particularly of the eyelids and lips. Occasionally visual disturbances or permanent blindness. Skin dry and hot. Full bounding pulse. Stertorous respirations.

Treatment: Morphine at once. Absolute rest. Head and shoulders to be somewhat elevated. (Oxygen may be of help)

Prognosis: If death does not supervene at once, recovery is probable.

III. Blast Injury:

Cause: Impact on chest wall of wave of positive air pressure following bursting of a high explosive. There results, if the injury is a severe one, (1) multiple hemorrhages in the lungs, most pronounced along their anterior borders near the mediastinum; and (2) loss of pulmonary elastic recoil.

Signs and Symptoms: Chest sometimes bulging. Normal percussion note but distant breath sounds. Bloody froth at mouth or nostrils. Shallow prolonged respiration; restlessness; prostration and grave shock. Surgical abdomen often simulated. Roentgenograms in conjunction with symptoms characteristic; scattered areas of patchy pneumonitis.

Treatment: Morphine for restlessness, despite signs of asphyxia. Fowler's position to improve breathing. Oxygen but not artificial respiration. Intravenous fluids or whole blood transfusions *contraindicated.* Administer concentrated blood serum if necessary. Venesection frequently averts lung edema.

Prognosis: Death may occur instantly. If one survives the first few hours, pulmonary edema developing, perhaps days later, may prove fatal. Therefore, it is important to minimize intravenous fluids.

FIRST-AID TREATMENT

At the moment the chest is injured, anxiety and restlessness develop. Cardiorespiratory embarrassment is shown by some degree of dyspnea and cyanosis. Often there is pain, seriously limiting respiration. Morphine in comparatively large doses should be administered at once. The patient should be kept warm. Elevation of the temperature above normal by excessive use of blankets or hot water bottles must be avoided, as peripheral vasodilation and loss of body fluids will result. These will add to the shock or throw a patient into shock if it is impending.

Unlike severe injuries elsewhere, the patient, if conscious, should be propped up to a semisitting position. He will then breathe more easily, and blood and mucus entering the bronchial tree may be promptly raised. If unconscious, he should be placed in the Trendelenberg position, so that blood and pharyngeal secretions will not be aspirated. If the dyspnea is the result of pain limiting inspiration, or if a crushing injury exists, after giving an adequate amount of morphine, adhesive strapping of the chest as described under "Stove-in Chest" may afford relief. Open sucking wounds of the thorax should be covered at once. Tension pneumothorax may be temporarily relieved by placing an open needle in the chest to prevent the accumulation of too much air. These conditions will be discussed later.

SURGICAL TREATMENT

The following emergencies, as encountered at the place where injury occurred, urgently require special and immediate attention.

I. "Sucking Wound" of Chest Wall:

Normally, during inspiration the ribs move outward and upward, the

sternum forward and upward, and the diaphragm downward. Thus the chest enlarges in all diameters. The volume of the lungs is proportionately increased by passively following the movements of the thoracic walls. The negative pressure thus created is at all times satisfied by the inrush of air by way of the trachea, which is the only communication with the outside air.

When a chest wall injury extends through the parietal pleura into the pleural cavity (normally only a potential space), two openings are present to admit air into the thorax. While on inspiration air enters the chest through both of these openings, it is only by way of the trachea and bronchi that air, with its necessary oxygen, can reach the pulmonary alveoli. It is evident that the percentage of air that reaches the lungs through the trachea is in inverse proportion to the size of the chest wall opening. When this differential is sufficiently great, not enough oxygen is available to sustain life even with the deepest inspiratory effort. The prompt application of a reasonably air-tight dressing to close the chest wall opening averts this disaster.

On arrival at the hospital, the dressing applied to the sucking wound of the chest wall is removed. Débridement is painstakingly carried out, using a scalpel, not a scissors. A scissors squeezes as it divides, while a sharp scalpel cuts cleanly. A bit of muscle or fascia, whose nutritional future is hanging in the balance, might by this slight additional injury eventually become necrotic. The main purpose of this procedure is to excise all devitalized tissue.

The divided parietal pleura should be snugly closed if possible. However, this is rarely possible, so that the chest wall muscles or even the lung may be sutured across the pleural aperture. If this appears unsafe or impractical, one may be forced to resort to tight gauze packing as a temporary life-saving measure. Closed pleural drainage, using a thumb-sized tube, is then established as described under "Empyema—Closed Drainage."

After careful débridement and after the parietal pleura has been closed, all raw surfaces are sprinkled with 4–8 Gm. of a sulfonamide. Because of the danger of infection no sutures are taken in the chest wall other than those necessary to close the pleura. The wound is then packed open with plain gauze. Rubber tissue applied directly over the open wound will prevent the gauze dressing from becoming adherent to the raw tissues (even vaselined gauze is painful to the patient on its removal). Powdering the wound twice daily with the sulfonamide is desirable. Healing time may be shortened by the application of adhesive strips upon the skin, to loosely approximate wound edges.

II. Tension (or Pressure) Pneumothorax:

A wound of the chest wall penetrating the parietal pleura, uncomplicated by injury to the lung, produces a pneumothorax. The closure of the chest wall opening, either by suture or dressing, effectually eliminates further

entrance of air. Subsequent aspiration of air before the injured arrives at the hospital is seldom necessary.

If, however, a laceration of the lung exists, complicating a penetrating wound of the chest, closure of the chest wall opening will remove but one source of the air accumulating between the lung and the chest wall. Bronchial branches torn open with the lung laceration allow air to escape through the torn visceral pleura with every inspiration. Almost always a check-valve mechanism at once occurs. That is, air enters the pleural space in inspiration by way of a bronchus in the lacerated lung, but in expiration it is impeded from escape because the lung tissues immediately surrounding the pleural opening are pressed together. Thus, a certain amount of air is retained. With each succeeding respiratory cycle the amount of imprisoned air increases. Air tension or pressure in the pleural cavity steadily increases. From the very moment of its inception, several important cardiorespiratory disturbances are initiated. The lung on the involved side is more and more collapsed. At the same time the mediastinum is pushed toward the opposite side at the expense of the contralateral lung, which, in turn, is steadily reduced in size. In the meantime, cardiovascular function, always intimately associated with respiratory disturbances, is becoming seriously affected. Unless relief is forthcoming, death supervenes. Immediate needle decompression, with or without syringe aspiration is life-saving.

Lung laceration with bronchial communication into the pleural space may occur following crushing injuries of the chest without any break in the continuity of the chest wall.

The diagnosis is not difficult. Dyspnea is of course the outstanding symptom. The side of chest containing the bronchopleural fistula is immobile. Percussion note is high. Breath sounds are absent.

Treatment: Immediate introduction of a needle, preferably of large size. This should be left in place, anchored by adhesive straps, until patient arrives at hospital. The needle may be inserted in 2nd or 3rd interspace just lateral to midclavicular line.

Warning: Do not needle indiscriminately left basal pneumothorax pockets as acute dilatation of the stomach or diaphragmatic herniation (discussed later) may be present.

On arrival at the hospital, if there are signs of accumulation of air in the affected pleural cavity, then the needle must be connected to an air-tight system under water, as depicted on the film. An air-tight intercostal catheter may be inserted into the pleural cavity and connected to a closed water bottle system in place of the needle. This has the advantage of allowing the escape of larger amounts of air and of offering less chance of trauma to the expanding lung, but has the disadvantage of presenting a greater chance of infection.

III. Cardiac Tamponade:

Following injury to heart muscle or of intrapericardial vessels, hemorrhage into the pericardial sac may occur. As bleeding progresses, space for cardiac

action is steadily lessened. The auricles and vena cavae and finally the ventricles are compressed. The venous pressure rises. The arterial pressure falls. Inadequate oxygenation of circulating blood results. Serious tamponade of heart exists.

The diagnosis, when the possibility of its presence is considered, is not difficult, nor, fortunately, is the procedure for its temporary relief particularly complicated.

Duskiness of mucous membranes and nail beds or cyanosis is present; moderate shortness of breath; dyspnea or stertorous respirations, depending upon degree of tamponade; small thready pulse; low arterial pressure; high venous pressure; engorgement of veins of neck; sometimes venous pulsation. Cardiac pulsations are faint or absent over the pericardium. Cardiac dullness is not necessarily much increased.

There is usually a time-interval between the moment of actual injury and the onset of unconsciousness because of cardiac tamponade. The reason for this is found in the fact that it takes time for sufficient blood to leak out into the pericardium to actually tamponade the heart. As the tamponade becomes marked, the low arterial pressure and marked venous stasis result in serious interference with the oxygen supply to the brain.

If the imminent danger of immediate death does not contraindicate additional diagnostic measures, fluoroscopic examination will usually establish, with certainty, the diagnosis when the findings are considered in conjunction with the history, symptoms, and physical findings. One sees a cardiac shadow, but little larger than normal, with little or no evidence of pulsation.

Immediate aspiration of blood from the pericardium is imperative. The site of election is the left costoxiphoid angle. By this approach the needle not only avoids passing through both pleural leaves, but also cannot lead to injury of the lingula of the upper lobe of the lung were it adherent to the pleura in the precordial region. Furthermore, by this subcardiac approach the needle will enter the pericardium below the heart and not directly toward it, as would occur if aspiration was attempted through the chest wall. Finally, the pericardial sac can be more satisfactorily cleared of blood when aspiration is carried out from below.

After tissues are anesthetized with 1% novocain from the skin to pericardium, the aspirating needle (No. 15 gauge preferably) is inserted, directing it inward and slightly upward. The bevel on its distal end should be comparatively short. Usually the passage of the needle through the pericardium into the less resistant blood within the sac is easily appreciated as the needle advances. The larger the syringe attached to the needle, the less tactile sensation is retained. Since the pressure of the blood within the pericardial sac is positive, it is often advantageous to insert needle with no syringe at all, so that maximum appreciation of the resistance of the various structures (especially the pericardium) with which it progressively comes in contact may be recognized. Blood should escape at once when the pericardial sac has been entered. Then the syringe, with two-way valve, is at-

tached and all blood possible is aspirated under air-tight conditions. At all times during the procedure great care must be taken to avoid needle contact with heart muscle. The constant motion of the heart transmits along the needle, to the one who is aspirating, a scratching sensation. The needle must immediately be slightly withdrawn, for even superficial injury to heart muscle may result in prolonged oozing of blood from the cardiac abrasion, sufficient to produce tamponade in its own right, some time after needle has been withdrawn.

When the diagnosis is made before the patient has been taken to the hospital, aspiration should be accomplished immediately if patient is in serious condition and the needle and syringe are available. Even the withdrawal of 75 to 100 cc. of blood may be temporarily life-saving. When, after thorough evacuation of the blood from the pericardial sac, signs of cardiac tamponade recur, surgical exploration for possible suture of the heart muscle or removal of foreign body is indicated.

The technic of intrapericardial exploration and suture of heart wound fall outside the scope of this discussion. It is shown in detail in the films on "Chest Injuries."

IV. "Stove-In Chest":

The injury consists of multiple rib fractures, often on two planes, anteriorly and laterally or anteriorly and posteriorly, usually limited to one hemithorax. The crushed side, because of its loss of rib support, cannot enlarge with inspiratory effort. The negative pressure created by the enlargement of the uninjured side of the chest in inspiration pulls the mediastinal organs toward the good side. The unoxygenated air in the lung of the injured side is actually sucked into the functioning one. In expiration both of these movements reverse. It follows then that the "stove-in" side of the chest actually decreases in size during inspiration, with a relative increase in size on expiration as the mediastinal organs return to midposition. The same air may move back and forth from the paradoxically moving lung to the good lung. The amount of air that goes back and forth between the lungs depends on the amount of paradoxical movement of the chest wall and the amount of mediastinal shift. This most inefficient type of respiration produces marked dyspnea. Severe pain on breathing that prevents even normal depth of inspiration may still further lessen oxygen supply.

Treatment: (1) Give morphine in large enough doses to control pain; (2) strap chest with a completely encircling wide adhesive bandage at the level of the costal margin as described in the next paragraph; (3) aspirate retained secretions in the trachea by catheter or, if necessary, through bronchoscope; and (4) establish at once and maintain a high sulfonamide blood level to combat pneumonitis.

Adhesive placed at the costal margin level should completely encircle the chest when the diaphragm is involved, either directly or indirectly. If there exists contusion of the diaphragm, a diaphragmatic pleurisy or injury to the intercostal nerves whose anterior branches are in the vicinity of the

diaphragm, splinting of the affected diaphragm is desirable. The diaphragm however is a bilateral organ. In order to limit the excursion of either side, both must be splinted. An adhesive bandage completely encircling the chest is required, and proves of great benefit.

When adhesive so applied is not well supported, then it should be removed, replacing it with a tight adhesive band applied in expiration around the injured side of the chest. It should extend three inches onto the opposite hemithorax, both anteriorly and posteriorly.

Paradoxical motion of the chest wall may be greatly decreased by various mechanical contrivances. The Drinker respirator, or similar type, if available, is the best device, as it completely does away with the need for a firm chest wall until the ribs have knit sufficiently to offer strong support. If a respirator is not available, simpler measures may be employed. Several towel clips can be snapped through the soft tissue and under the ribs. On attaching the towel clips to a fixed overhead support, the depressed chest wall may be maintained in an elevated position.

V. HEMOTHORAX:

The presence of a considerable amount of fluid between lung and chest wall produces characteristic signs, whether it be serous effusion, pus, or blood.

There is evident decrease in the mobility of the injured hemothorax. The degree depends upon the amount of blood present in the pleural space. The involved side often appears to be larger than the contralateral one. The percussion note is flat over the hemothorax. Tactile fremitus is absent. Breath sounds are usually absent. Pure bronchial breathing may be heard if the layer of blood is so thin that the breath sounds in the collapsed lung are transmitted through the blood to the chest wall.

When the lung is injured, in addition to the hemorrhage, air escapes from the bronchi. The presence of this air rising through the blood causes the percussion note to be tympanitic rather than flat. The signs then are predominately those of pneumothorax. The indications for relief in the field are those discussed under pneumothorax. On arrival at the hospital a roentgenogram will disclose the presence of the blood. If a film with the patient in the sitting position can be taken, without undue hazard, a fluid level establishes the diagnosis. If not advisable to raise the patient, a chest film with patient supine will show haziness—indicative of pleural fluid lateral to the lung, in a field that would be clear were air alone present.

First-aid treatment follows the general rules mentioned above—morphine, absolute rest, external heat. Aspiration of blood, except under rare circumstances, is contraindicated before arrival at the hospital. If death occurs during this period, it is usually from exsanguination, not from the pressure of extravasated blood against the mediastinum.

At the hospital, early aspiration of blood is desirable (24 hours after injury and before the 4th day).

Caution: If an X-ray is not available, or the diaphragm has not been well visualized roentgenographically, before aspirating the left chest insert

the needle at the level of 5th or 6th intercostal space so as to avoid puncture of the diaphragm. Conceivably, the pressure of the blood against the injured lung has occluded the vessels responsible for the hemothorax. Premature reexpansion of the lung by the aspiration of the extravasated blood in the pleural space might cause bleeding to recur. To obviate this contingency, therefore, the blood aspirated during the first few hours after injury should be partially replaced with air. Blood removal appreciably shortens convalescence and permits the recognition of continued bleeding, also, whether or not the lung concealed by it is atelectatic. Extravasated blood produces extensive pleural irritation in some patients. Its early removal greatly reduces the danger of progressive pleural fibrosis. Furthermore, aspiration of the blood with partial replacement of air lessens the size of the pleural space should infection eventually develop.

If large clots are present in considerable amount, aspiration is not successful. It is frequently advisable to open the chest, remove clots, rinse out the pleural space with saline, sprinkle pleural surfaces liberally with a sulfonamide powder, and close the operative wound. The air remaining in the pleura after tight chest wall closure should be at once aspirated until a negative intrapleural pressure to five or ten centimeters of water be obtained. Higher pressures exerted on a lung resistant to sudden reexpansion because of thickened visceral pleura, may cause an outpouring of serum to satisfy the partial vacuum created by the aspiration of air. Subsequent removal of air in comparatively small amounts at daily intervals is the preferable procedure.

VI. Infected Hemothorax:

Even when the blood in the pleural space is sterile, a daily temperature rise to 100°–101° F. is usual. When, after several days there is an increase in temperature, pulse, and respiration, infection should be suspected.

The following findings point to an infected hemothorax: (1) Increased toxicity; (2) evidence that the amount of fluid in the pleural cavity is increasing (outpouring of serum from pleural irritation); and (3) the sample of blood aspirated has become purplish in color or has an unpleasant odor.

Bacteria often develop, at first only at some certain spot in the bloody fluid. So it may be several days before bacteria are prevalent throughout all the blood. The presence of purplish blood of unpleasant odor in an increasingly toxic patient is exceedingly suggestive, even though the culture of the blood aspirated is sterile. The complete and immediate removal of all the old blood, with adequate tube drainage of the pleural cavity (closed method) is highly desirable. The technic of pleural drainage is discussed elsewhere in the paper under "Empyema."

VII. Penetrating Wounds of the Chest:

The mortality rate following present day technic in intrathoracic operations is very low. In consequence, surgical exploration of intrathoracic injuries is much more frequent in the present war because:

1. Foreign bodies can be removed. The presence of phosphorus in

incendiary bombs makes retention of even comparatively small bits of metal from them hazardous.

2. In injuries both of the lung and chest wall, devitalized tissue can be débrided and the injury repaired. Openings in the lung may be closed in some cases.

3. Bleeding vessels of the chest wall and lung can be ligated. Injury to the internal mammary and intercostal vessels may lead to serious intrapleural hemorrhage. Their ligation may be life-saving.

4. Blood and clots between the pleural leaves or a superficial hematoma of the lung can be removed.

5. The diaphragm can be examined as to evidence of injury to it. If an opening through the diaphragm is found, it is enlarged so that the intra-abdominal organs immediately below may be examined. By these procedures the danger of subsequent infection from bits of clothing, torn lung, *etc.*, may be greatly reduced, recovery hastened, and early or late death from hemorrhage, or sepsis averted.

Before decision is made to explore, shock must be controlled. If possible, roentgenograms of the chest with patient in upright position to show fluid level, should be obtained. Air replacement of blood in the pleural cavity is of great diagnostic importance for reasons previously mentioned.

VIII. Traumatic Atelectasis and Pneumonitis:

Cause: The most important factor is the impairment in efficiency of the cough mechanism with the subsequent retention of bronchial secretions. It may be produced by the following conditions: (1) Voluntary splinting of the painful chest wall; (2) paradoxical movement of the chest wall; (3) oversedation; and (4) prolonged unconsciousness. Keeping the patient in one position causes hypoventilation of the lungs, while pulmonary hemorrhage may actually plug a bronchus and so directly cause atelectasis.

Diagnosis: The presence of retained secretions may be detected by finding coarse râles over the trachea and large bronchi. Cyanosis, fever, dyspnea progressing to coma, with concomitant chest signs, point towards an atelectasis or traumatic pneumonitis. There is usually dullness to percussion. Breath sounds, as a rule much decreased, vary from vesicular to bronchial. Râles are usually present over the affected area. The roentgenograms of the chest are diagnostic, showing changes varying from a scattered, patchy infiltration to a complete lobar haziness, with or without mediastinal shift, elevated diaphragm, or narrowed interspaces. Any fever in a patient with a chest injury should be considered as caused either by atelectasis or traumatic pneumonitis, unless proven otherwise.

Treatment: The prophylactic treatment is the more important. This includes supporting of the thoracic wall by adhesive taping and the use of shot-bags, and encouraging the patient to cough by the manual holding of the chest wall, which lessens the chest pain. Enough morphine should be used so that pain does not prevent the patient from coughing. Over-

doses of opiates or barbiturates that result in long periods of coma or semi-stupor, when the patient cannot cough voluntarily, are to be studiously avoided. Ten per cent carbon dioxide (not carbogen) is an important aid. The patient should be turned from side-to-side every two hours if possible, and expectorants and inhalants utilized to make the sputum less tenacious and hence more easily raised. A high blood sulfonamide level should be established early in all cases of chest trauma where injury has been severe enough to produce atelectasis or traumatic pneumonitis.

Catheter suction of the trachea and bronchi is, in most instances, the procedure of choice, to aspirate retained secretions and blood. The convenient technic is that of inserting a No. 16 urethral catheter through the nares and advancing it through the larynx on deep inspiration. The simplicity of this method is shown on the film, and can be mastered after a little practice. Local anesthesia produced by spraying the throat with two per cent pontocaine, will facilitate the passage of the catheter into the larynx, especially in the presence of an active gag reflex. Similarly, spraying the nasal mucosa with pontocaine lessens the discomforture to the patient. There is no contraindication of local anesthesia to the throat if fluids by mouth are withheld until the gag reflex returns (two hours). The catheter is attached to an ordinary "nose and throat" type of suction device capable of producing 15 pounds of pressure. The catheter may then be advanced down the trachea. By turning the head to the right side, the catheter may be introduced into the left main bronchus, and by turning the head to the left side, after withdrawing the catheter a little, it may be introduced into the right main bronchus. The suction should be applied intermittently so that too much air is not completely drawn out of the bronchial tree at one time. Cough is stimulated by the irritation of the catheter on the tracheal and bronchial mucosa so that frequently more sputum is coughed up around the catheter than is aspirated through it. This procedure has the advantage of being readily available, of being simple, and may be quickly set up and instituted, and used repeatedly on one patient. We believe too little attention throughout the country has been paid to this simple and highly efficacious procedure.

Bronchoscopy, of course, is more thorough, and should be employed when it is impossible to pass the catheter or where a thick mucous plug or blood clot resists removal by the catheter. It should be undertaken in preference to catheter suction where foreign body is a possibility or vomitus has been aspirated. The trained bronchoscopist can easily perform this procedure (as shown on the film) without moving the patient from his bed. The fact that the patient is critically ill is not a contraindication to bronchoscopy when atelectasis is present, as this condition may be the principal cause of the precarious state of the patient. Bronchoscopy in bed causes no more serious disturbance to the patient than that resultant from hard coughing, a respiratory exercise that usually is most desirable.

IX. Subcutaneous Emphysema:

Following chest injuries both penetrating and nonpenetrating, bronchial communication may occur between torn lung and subcutaneous tissues.

Swelling and crepitation beneath the skin indicate the site of subcutaneous air. While gas-forming bacteria may be responsible for the presence of air beneath the skin, the absence of toxic symptoms, of edema, and of dark red or coppery discoloration of skin, render the latter source of the crepitations untenable. If emphysema is steadily progressive until most, or all, of the body is involved, open operation near the site of injury to repair the injured lung and close the open bronchus may be indicated. It is seldom, however, that such operative measures become necessary.

X. Mediastinal Emphysema:

Cause:-Rupture of bronchus in or near mediastinum; rupture of bronchus deep in the lung with extension of air along the peribronchial tissues.

Symptoms: Subcutaneous emphysema first appears in episternal notch but may spread extensively under skin of head, trunk and extremities. There may be dysphagia, dyspnea, cyanosis, pneumatic extrapericardial tamponade, distension of the neck veins, and finally circulatory failure.

Treatment: Palliative transverse incision at jugulum through platysma. By blunt dissection with finger, opening may be extended beneath manubrium into mediastinum.

Curative: Attack surgically at source if condition of the patient is critical.

XI. Miscellaneous:

Acute dilatation of the stomach occasionally occurs at a most serious complication of crushing injuries of the chest, with or without phrenic nerve paralysis on the left. Vomiting, when present, especially if accompanied by distention or tympany over the upper abdomen, is highly suggestive. A left basal pneumothorax may be simulated. Even though these findings are present only in a slight degree, acute dilatation of the stomach must be suspected when the patient, becoming steadily worse, has a rising pulse and a falling temperature. The passing of a stomach tube, an extremely simple procedure, is life-saving.

Rupture of the diaphragm, with the herniation of the abdominal contents up into the chest cavity, may produce dramatic and alarming symptoms. Extreme dyspnea, vomiting, with the signs of fluid and air in the lower left chest may be present. Borborygmus, if demonstrable, is diagnostic. Needling of left lower chest must be postponed until diaphragmatic dome has been visualized roentgenographically. If the diaphragm cannot be identified on the plain film and barium is not available, the giving of soda bicarbonate by mouth, followed by a few drops of dilute hydrochloric acid will produce a gas bubble to readily identify the diaphragm.

The surgical repair of the diaphragmatic herniation should be postponed if possible until the patient has otherwise made a complete convalescence.

INFECTIONS OF THE THORACIC ORGANS FOLLOWING TRAUMA

With prompt débridement of open chest wounds, the use of the sulfonamides, and attention to the principles outlined earlier in this paper, many thoracic infections following trauma can be prevented. However, because of the exigencies of modern warfare it is often impossible for a large number of cases to receive adequate early first-aid or hospital treatment. If these cases survive the initial shock and hemorrhage, infection of the chest organs will be a common finding. In some cases, too, in spite of the most painstaking early care, infection will ensue. Therefore, no summary of the management of chest injuries is complete without a brief discussion of the common infections of the thoracic organs that follow trauma.

I. Infections of Soft Tissues of Chest Wall:

Infections of the soft tissues of the chest wall differ little from those elsewhere in the body, with one outstanding exception. The chest wall may become infected by anaerobic organisms from a putrid empyema or lung abscess, either through a needle track or direct extension of the inflammatory process. In either case a fulminating cellulitis develops that is often rapidly fatal. Prophylactically, the prompt institution of surgical drainage of the putrid empyema will avoid multiple and hazardous needlings through the chest wall.

The usual procedure, in the attempt to control a rapidly progressive cellulitis of the chest wall, has been to make long parallel incisions through to the floor of these infected areas, extending the incisions well into apparently uninvolved tissue on either side. The wound edges are widely undermined with the fingers and rubber drains inserted plentifully to hold the infected surfaces apart. Uniformly, these patients at operation are profoundly toxic. The surgeon, by the above method, frequently administers the *coup-de-grace* by ruthlessly breaking down whatever barriers the body has been able to erect against the invading bacteria.

We have found it much more satisfactory to make a wide incision in the uninfected tissue across the path of the oncoming bacterial invasion, an inch or more in front of the infected area. Usually the spread is extending in but two directions. Two such incisions should be all that would be required to halt this process. Vaselined gauze is placed in these zonal incisions to prevent, temporarily, apposition of the wound edges. As a rule, limitation of the infection is thus established. After these incisions peripheral to the spreading cellulitis are completed, one or perhaps two cuts are very gently carried through into the infected planes, avoiding any molestation of the surrounding tissues. Liquefaction, which usually occurs within a day or two, is easily controlled by multiple small incisions, with minimal danger of disturbance to surrounding tissues. Subpectoral and subscapular abscesses may be the source of occult fever during their early stages. Sooner or later local pain, tenderness, swelling, and fluctuation will point to the diagnosis. Wide drainage with the dependent placing of multiple rubber-dam drains

generally is effectual in draining these abscesses. If they persist over a period of time, one must be on guard for a possible osteomyelitis of an adjacent bone.

II. Osteomyelitis of Ribs and Sternum:

Osteomyelitis of the ribs practically always follows trauma and is rarely a part of osteomyelitis with multiple involvement of other bones of the skeleton. Simple fractures of the ribs are practically never complicated by osteomyelitis. However, osteomyelitis of the ribs does occur in infected compound fractures and when chest wall infections and empyema are present, if any bare rib deprived of its nourishing periosteum is exposed in the infected wound.

Diagnosis: The diagnosis is made by the history of local trauma, the signs of a low grade infection, and local tenderness. Roentgenologic examination almost always shows the erosion and periosteal proliferation typical of osteomyelitis.

Treatment: The treatment consists of wide excision of the involved bone down to healthy osseous tissue. If a cartilage is exposed, or infected it must be completely removed, otherwise permanent healing will not take place.

In a similar manner osteomyelitis of the sternum usually follows trauma. More constitutional reaction is present and there is local tenderness, swelling and redness. Although it is sometimes difficult to demonstrate the lesion, the roentgenograms show typical signs of osteomyelitis. Because the complications of retrosternal abscess and anterior mediastinitis are serious, wide excision of the involved bone and adjacent costal cartilages is indicated.

III. Empyema:

Empyema is the most common thoracic infection following chest injuries. This is true because of the high frequency of cases in which the pleura is soiled by penetrating wounds and because empyema is not an uncommon complication of pneumonia and lung abscess.

Diagnosis: Empyema that follows thoracic trauma is usually not difficult to diagnose. If there has been a penetrating wound, usually débridement and drainage has not been adequately carried out. There is a high septic-type of fever with corresponding elevation of pulse and rapid respiration. The physical signs are those of fluid, which often accumulates very rapidly. The roentgenogram shows signs of pleural fluid on the involved side. In the absence of gas-forming organisms, no fluid level will be seen unless there has been a tear of the lung or an open pyopneumothorax is present. The diagnosis is made by the aspiration of purulent fluid from the pleural cavity. Immediate smear and culture of the pleural fluid must be done, as often it is impossible to demonstrate the organisms after the material has stood overnight. Before drainage is undertaken, unless the odor of the pus is foul, pyogenic organisms must be demonstrated in the pleural fluid.

TREATMENT

1. *Open Drainage:* Open drainage with rib resection, under local anesthesia, and the insertion of a large tube is the procedure of choice. However,

certain conditions must obtain. In the discussion on "Sucking Wound of Chest Wall," the dangers of the open pneumothorax were emphasized. When the pus aspirated from the pleural cavity is thin, there is little fibrin content and, therefore, few adhesions are present along the mediastinal pleura. Hence, the mediastinum is mobile. Drainage at this time would subject the patient to the dangers of the open pneumothorax and possible fatal issue. Therefore, needle aspiration is the preferred treatment early in acute pyogenic empyema, not only so that the mediastinum will be fixed, but also that the size of the empyema pocket shall be limited. When the fluid contains 80–90 per cent sediment, open drainage with rib resection and the dependent placing of a thumb-sized tube, large enough to evacuate thick pus and fibrin, is the procedure of choice. If a pyopneumothorax is not present, 50–100 cc. of the pus aspirated may be safely replaced with air to produce a fluid level. Then with a lateral decubitus roentgenogram the bottom of the pleural pocket may be accurately determined. However, if such a roentgenogram is not available, it is safe to resect the rib immediately above the lowest intercostal space where pus was obtained by aspiration. We do not completely aspirate all the pus from the pleural cavity immediately before surgery, as it may be impossible to find the pocket in the operating room.

2. *Closed Drainage (Large Tube):* Because the lumen of an intercostal catheter is not large enough to permit the drainage of thick pus, fibrin, and blood clots, we feel that it is not indicated in empyema. We reserve this method (1) to drain uninfected bloody serum in clean operations; and (2) to allow the escape of air in tension pneumothorax, where the needle method of reduction of air pressure has not been successful. To effect closed drainage with a large tube, a six-centimeter segment of rib is resected, under local anesthesia, at the posterior axillary line. Providing the resultant drainage is dependent, this site is desirable as the patient does not have to lie on the tube. The bottom of the pleural pocket is determined according to the technic described under "Open Drainage of Empyema." Through an opening in the parietal pleura, just large enough to admit it, a thumb-sized tube is snugly inserted into the pleural cavity. This tube is then connected to a drainage bottle on the floor with an underwater seal. The soft tissues overlying the parietal pleura are not sutured but approximated by flamed adhesive strips applied to the skin. Thus, the air-tight closure is helped by this strapping, and yet infected secretions are not dammed up in the soft tissues. By using this method (depicted on the film) air-tight drainage is maintained, yet the use of a thumb-sized tube permits the drainage of thick pus and fibrin. This type of drainage following chest injuries is indicated in:

a. *Chest Injuries where Pleura is Grossly Contaminated:* Here, following débridement, because the mediastinum is not fixed, closed drainage is indicated. (The use of a large tube makes it adequate)

b. *Open Operations of the Thoracic Cavity where the Pleura is Grossly Contaminated—such as Lobectomy:* The tube in this case should be put in posteriorly to drain the hilum.

c. *Putrid Empyema,* if caused by a highly virulent strain of organism, should be drained early, because repeated aspirations lead to phlegmon of the chest wall. Since the mediastinum is not fixed *early,* closed drainage is necessary.

d. *Chronic Empyema—where Highly Negative Suction Therapy is Indicated:* Prompt, adequate drainage usually prevents the acute empyema from becoming chronic. However, if the drainage tube is too high, too small, or pulled out before the pleural space is obliterated, chronicity may result. The presence of foreign bodies, bronchopleural fistulae, tuberculosis, fungi, or osteomyelitis of the ribs, may also make the empyema chronic.

The Schede thoracoplasty is the time-honored treatment for this condition. Recently we have used marked negative intrapleural suction through the use of a large drainage tube. Although it is not always successful, it should be tried before the Schede thoracoplasty.

IV. Lung Abscess:

Lung abscess may follow penetrating or nonpenetrating wounds of the chest. When the patient is unconscious from shock on the battlefield, the aspiration of secretions from the upper respiratory track (particularly if oral sepsis is present) may be followed by pulmonary abscess. Bronchial obstruction caused by the aspiration of a foreign body or clots from pulmonary hemorrhage and less often septic emboli may also cause pulmonary abscess following injury to the chest.

Diagnosis: The diagnosis of lung abscess in a patient with a severe thoracic injury may be difficult, as other conditions may mask symptoms referable to the abscess. Chills and high temperature occur first, followed by a harassing dry cough that soon is productive of blood-streaked mucus. The raising of foul gas precedes the "vomica" or sudden coughing up of profuse amounts of purulent or foul sputum as the abscess breaks into the bronchus. Foul sputum is the most important diagnostic sign as it almost invariably means pulmonary destruction. Putrid bronchiectasis and the rupture of a putrid empyema into a bronchus are the two other main conditions resulting in foul sputum. The physical findings are notoriously deceptive. Rarely are the physical signs of cavity present in acute lung abscess. Dullness, diminished breath sounds, and medium to coarse râles are common findings. The roentgenogram is usually diagnostic, showing, at first, an area of dense "infiltration" and then a cavity with a fluid level. However, as long as the draining bronchus is blocked and the lung abscess cavity completely full, no fluid level can be seen. Care must be employed to take the roentgenogram in the upright position (to demonstrate a fluid level) just after the patient has raised considerable amounts of sputum, so that the level may be present.

Treatment: As soon as the diagnosis of lung abscess is made, medical measures should be at once instituted. In addition to sulfonamide therapy and a good hygienic regimen, indicated for all pyogenic infections, postural drainage (if with change of position cough not alarmingly violent) and bron-

choscopy are of first importance. The branch bronchus draining a lung abscess is always severely inflamed—leading to obstruction of this bronchus because of the edema and swelling of the mucosa. At bronchoscopy, shrinking solutions can be directly applied to the swollen mucosa. The pus may thus be aspirated as the drainage into the main bronchus is improved. Frequently during the first hours following bronchoscopy, expectoration of pus is particularly copious. Therefore, bronchoscopy, should be uniformly employed in pulmonary abscess. Because there exists a constant chance of spread of the infection to other lobes and because the lung abscess walls become fibrous after a time, the sooner the bronchoscopy is done the better. The clinical course, as shown by fever, toxicity, and amount of sputum, is important in evaluating the progress of the patient. However, the chest roentgenogram is the deciding factor. If the roentgenogram shows no *definite* diminution in the size of the abscess cavity after bronchoscopy, in spite of apparent clinical improvement, external drainage is indicated. It is a prevalent medical opinion that after the diagnosis of lung abscess is made, one to two months should elapse to allow the patient to heal spontaneously. With the present day technic of surgical drainage under local anesthesia, as shown on the authors' films dealing with "Chest Injuries," the percentage of cures is so high, and the mortality so low, that this unnecessary delay is unwarranted. Time does not permit us to cite the evidence for external drainage. Yet our results of early surgical drainage have been so good that we can categorically state, that when a lung abscess stops improving and becomes stationary in its course, constant danger of spread exists and drainage is at once indicated.

The postoperative care is important and entails constant vigilance to make sure that the pulmonary pocket is healed before the chest wall sinus is allowed to close. Because of success in closing the pulmonary pockets with fat grafts (technic illustrated on film), we are using them almost as a routine measure to shorten the otherwise often prolonged convalescence of the patient.

V. Suppurative Pericarditis:

Diagnosis: Suppurative pericarditis follows penetrating wounds and foreign bodies of the pericardium. It is also secondary to pneumonia and empyema. In addition to the general signs of marked sepsis, enlarged precordial dullness, the signs of cardiac tamponade described earlier, and a palpable tender liver are present. The heart shadow is diffusely widened at the apex and base. Aspiration of purulent material by a needle in the costoxiphoid angle, as depicted on the film, is necessary to make the diagnosis.

Treatment: The treatment consists of general supportive measures, sulfonamides intrapericardially, and surgical drainage as soon as pyogenic organisms are demonstrated in the pericardial fluid on smear or culture. A parasternal incision, with resection of the entire 5th cartilage, or an incision parallel with and beneath the costal margin may adequately drain the pericardium without opening the pleural or peritoneal cavities.

VI. Mediastinitis:

Diagnosis: Rupture of the esophagus or trachea, whether from actual

perforation or due to a nonpenetrating wound, is the most common cause of mediastinitis accompanying thoracic trauma. Infected foreign bodies in the mediastinal tissues present another important cause of acute mediastinitis in persons with chest injuries. The breakdown of suppurative mediastinal lymph nodes or the spread of infections from adjacent organs also results in mediastinitis. The pus which is located in either the anterior or posterior mediastinum, may break out through the intercostal spaces or follow the fascial planes to the neck or extend downward between the pillars of the diaphragm.

The signs and symptoms are those of a fulminating inflammatory process plus the effect of pressure on the mediastinal structures. Frequently the general constitutional reaction is so severe as to mask the focal symptoms. Infection in the anterior mediastinum results in pain under the sternum, dilatation of superficial veins, dyspnea, respiratory stridor, tenderness on pressure on the sternum. If the posterior mediastinum is involved, pain between the shoulder blades, dysphagia, dyspnea occur. Dullness in the paravertebral region or increased anterior mediastinal dullness may be present. The roentgenogram generally shows a widening of the anterior or posterior mediastinal shadows. Air may be present. A fluid level helps in the localization especially if, lateral, oblique, and decubitus films are taken.

Treatment: In addition to general supportive measures and sulfonamide therapy, the treatment consists of early surgical drainage. If the abscess is in the anterior mediastinum, a paramediastinal incision is made, with complete removal of the incised cartilage to prevent certain chondritis of the remaining stump. The pleural space is avoided and the mediastinal pocket entered. If a posterior mediastinal abscess is above the fourth rib, it may be drained in the cervical region, entering at the anterior margin of the sternocleidomastoid muscle. Below the level of the fourth rib, resection of mesial segments of two or three ribs along with their corresponding transverse processes at the appropriate level, will lead to the exposure of the abscess cavity. The pleura is retracted laterally and soft rubber tubes, rubber dam, or gauze is inserted. Since the mortality without surgery is 80 per cent, and that with surgery is 30 per cent, early operation is recommended for this desperate condition.

VII. Bronchiectasis:

Bronchiectasis is such a late sequela of thoracic trauma that it is inserted here only for completeness, and warrants only the briefest remarks. Bronchiectasis follows a long-standing obstruction of the bronchus such as an undetected foreign body in the bronchus. It is also a complication of chronic lung abscess and suppurative pneumonitis. If the infections discussed earlier in the article are promptly and thoroughly dealt with, few cases, indeed, will end up with bronchiectasis. When any of these cases discussed above drag out, with chronic cough and expectoration, or repeated hemoptysis, bronchiectasis should be suspected.

The diagnosis is made only by outlining the bronchial tree with lipiodol dropped into the trachea. Bronchoscopy, however, should be always performed to rule out foreign body, granuloma, or tumor. Palliative measures

such as postural drainage and repeated bronchoscopies are of some benefit. If the bronchiectasis is sufficiently localized, lobectomy and pneumonectomy (as shown on the film) are curative.

SUMMARY

Approximately 25 per cent of the patients with thoracic injuries die between the field and the collecting station. The treatment of chest injuries should, therefore, take precedence over those of other parts of the body. An exception is the control of hemorrhage. Since shock may be directly due to serious cardiorespiratory disturbances it is often of vital importance that the chest emergency be cared for first. The cause removed or alleviated, shock may cease to be of moment. A thorough knowledge of the signs and symptoms, previously explained in this discussion, particularly those of cardiorespiratory disturbance, is, therefore, necessary. Once the diagnosis is established the immediate and perhaps urgently required treatment is as a rule comparatively simple:

1. The Closure of a Sucking Wound of the Chest Wall.
2. The Aspiration of Air to Relieve a Pressure Pneumothorax.
3. The Aspiration of a Cardiac Tamponade.

Even upon arrival at the hospital, open operations into the chest are not to be lightly undertaken. In the most skilled hands great, and sometimes disastrous, complications may be encountered. If there appears no clear purpose for surgical exploration it is wise not to explore until some definite indication does present itself. It is particularly important that the anesthetist be trained in tracheal intubation, and that adequate facilities are provided for positive pressure anesthesia. Seldom, indeed, is there justification in attempting a procedure of any magnitude before the injured has reached a hospital.

"Reparative Surgery" that can be postponed without undue hazard should be delayed until the condition of the patient has improved sufficiently to permit it. Infection must be fought by the prompt establishment of a high sulfonamide blood level and by early surgical drainage following the principles discussed in this paper.

The authors wish to express their sincere appreciation for the valuable suggestions made by Doctors Evarts A. Graham, Frederick Coller, Leo Eloesser, John Alexander, Edward D. Churchill and Isaac Biggers in the preparation of the film and this discussion.

PILONIDAL CYSTS AND SINUSES: A METHOD OF WOUND CLOSURE*

REVIEW OF 230 CASES

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A PILONIDAL SINUS OR CYST, known in the European literature as a sacrococcygeal sinus or cyst, is ordinarily thought of as a minor ailment and few clinics have devoted any particular care or study to the disease. As a rule its operative treatment and after-care are left to the less experienced members of the surgical staff who understand thoroughly the need of complete eradication, but often do not appreciate the importance of the subsequent treatment of the wound. With the best of care, the postoperative wound may require weeks and even months to heal. From the civilian point of view the time of actual disability is not long, but the extended period of dressings is a nuisance to the patient and to his physician. In the Army, on the other hand, a soldier must remain in the hospital until the wound is completely healed and he is able to return to full duty. This is often a matter of several weeks, or even months, and during this time not only the soldier's services are lost but he also becomes a liability to his government. A shortening of the healing period, therefore, is obviously desirable.

Recognition is usually given to Jonathan M. Warren,[1] of Boston, as the first to have called attention to this disease. In 1854, he described an "Abscess Containing Hair on the Nates," which undoubtedly was an infected pilonidal sinus; in 1867, he[2] reported further experiences with the condition and gave an excellent clinical description of the disease as we know it today. Credit for the first report of a case, however, apparently should go to A. W. Anderson,[3] also of Boston, who, in 1847, told of having extracted hair from an ulcer in the sacrococcygeal region.

As the disease became better known, considerable speculation naturally arose as to its etiology. There have been two principal theories concerning its origin, both of which concede its congenital nature.

One theory explains these epithelial sinuses and cysts as being derived from imperfect involution of the epithelial cells that participate in the formation of the embryonal medullary canal and its surrounding medullary, or neural, tube. Normally, the caudal portion of the medullary canal and tube become almost completely obliterated during intra-uterine life and are represented at birth by the filum terminale which extends from the distal end of the spinal cord to the back of the first segment of the coccyx.[4, 5] It is conceived that epithelial cells of the caudal end of the neural tube may sometimes persist to adult life and, as epithelial rests, give rise to

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pilonidal sinuses and cysts. This theory was advanced by Tourneau and Herrmann,[6] in 1887, and has received considerable support, notably from Mallory,[7] and Gage.[8] Further evidence tending to confirm a developmental relationship between pilonidal sinus and the cord is afforded by Moise,[9] and by Ripley and Thompson,[10] who reported cases in which sinuses in the sacrococcygeal region became the route of infection for meningitis.

According to a second theory, pilonidal sinuses and cysts are derived from the skin through a process of invagination, which takes place during intra-uterine life. In describing the case of a young girl with multiple congenital anomalies Féré[11] referred to what appears to have been a pilonidal sinus as a cutaneous infundibulum of the sacrococcygeal region. Stone[12] apparently accepted the theory of cutaneous invagination but was at a loss to explain why this inversion takes place. He[13] later conceived the idea that this development in the human was comparable to the invagination of skin structures seen in the preen gland of birds and represented an atavistic tendency in man. Fox,[14] basing his opinion on embryologic studies, was thoroughly convinced that pilonidal sinus is a derivation of skin ectoderm and not neurogenic or enteric in origin. Kooistra,[15] after sectioning a number of embryos, found evidence to support both theories, and the matter still remains unsettled.

There is some indication that heredity may be a factor, though probably a small one, in the occurrence of pilonidal sinuses. Cases are sometimes found in more than one member of a family, and their presence in identical twins has been reported by Mechling,[16] and by Goldberg and Bloomenthal.[17] The disease is occasionally associated with other congenital anomalies, as in the case reported by Féré,[11] but this is by no means the rule. In most of the large series reported there are one or more instances of spina bifida, but this probably is incidental.

Pilonidal sinuses are preponderantly a disease of young adult males and, therefore, become a matter of some military importance at this time. The ratio of males to females is usually reported as something like three or four to one. In the series to be herein reported the ratio is approximately three males to one female. Newell,[18] however, reported a predominance in the female. Some idea of the general incidence of the disease may be obtained from Kooistra's[15] finding that a diagnosis of pilonidal sinus was made on 350 of 313,285 patients admitted to the University Hospital, Ann Arbor, during a period of 14 years, an incidence of approximately 1:940. Seventeen of the 350 were recurrent lesions. As a rule the sinus or cyst gives no trouble before puberty. Attempts have been made to directly relate the onset of symptoms to the development of the sex glands, but there appears to be little evidence to support this thesis. It is probable that the development of hair which occurs at this period has something to do with the production of irritation in the sinus, but this assumption is open to question because not all pilonidal sinuses are hair-bearing. In Kooistra's[15] series 52 per cent contained hair, and in the cases reported by Smith[19] hair was found in only 40 per cent.

The disease appears to belong almost exclusively to the white race. A few cases in the Negro have been reported but Kooistra[15] states that no cases have been reported in the yellow, brown, and red races. The people of these races have relatively little hair on the body and this may be one factor in the absence of pilonidal sinus disease. Our series of 214 patients is made up of 55 Italians, 47 Central Europeans, 41 Jews, 24 Anglo-Americans, 16 Irish, 8 Poles, 5 Greeks, 5 Russians, 3 Armenians, 2 Negroes, 1 Spaniard, 1 Maltese, 1 Finn, 1 Turk, and 4 of undetermined origin. Considering the mixed population of the City of New York the racial distribution of cases is probably of small statistical value.

There is some difference of opinion as to the importance of trauma in the activation of a pilonidal sinus. Thirty-two per cent of Kooistra's[15] 350 cases gave a definite history of injury, but 87 per cent gave no history of injury within a month of the onset of symptoms. Of our 214 patients, only 13, or 6 per cent, attributed the onset to direct injury. In addition to this number it is interesting that five of the 57 female patients in the series, or 8.8 per cent, gave childbirth as the cause of onset and two others stated that symptoms began during pregnancy. Kooistra[15] was of the opinion that injury is a relatively unimportant factor in the etiology of pilonidal sinus, and in this he is supported by Breidenbach and Wilson,[20] and others. In military surgery, however, one is impressed by the number of cases coming from the mechanized units where scant cushioning of metal seats is the rule.

Fundamentally, the lesion consists of a single sinus or of multiple midline sinuses leading to one or more cystic cavities situated in the subcutaneous fatty and fibrous tissue overlying the region of the lower sacrum and coccyx. The external opening is situated caudal to the cyst and a probe entering it passes obliquely in an anterior and cephalic direction. According to Rogers,[21] 70 per cent of the sinus tracts are of this simple variety. He found no evidence that the epithelial rests constituting the original congenital defect had any tendency to extend except as they were forcibly displaced by abscess and scar tissue formation. David[22] is also of the opinion that the complications of epithelial arrangement are caused by the development of an abscess within the cyst which upon rupturing into the surrounding tissues carries with it a part of the cyst wall. The displaced portion of cyst wall gradually forms a new cavity lined with epithelium. Whatever the explanation may be, it is well known that some of the tracts do have ramifications.

The great majority of cases have some degree of infection and, as a rule, there is a history of abscess or of drainage from the sinus. Infection was present in all of Roger's[21] cases. The same was true of virtually all of the 214 patients comprising the series being reported. Exactly one-half of the 214 gave a history of one or more abscesses which had either been incised or had ruptured spontaneously.

The symptoms and signs of the disease are too well known to warrant extended discussion. They are due almost entirely to the infection, which

differs clinically from ordinary infections by persisting for a long time after drainage has been established. The infection rarely gives rise to a bacteremia, but in one of our cases arthritic symptoms appeared to be definitely related to exacerbations of the infection in the sinus.

Differentiation of this disease from other conditions which manifest themselves in the same region is usually not difficult. Occasionally the opening of a sinus near the tip of the coccyx is mistaken for an anal fistula, but the careful introduction of a probe or a roentgenogram following the injection of radiopaque medium will as a rule establish the diagnosis. An infected sinus or cyst may be mistaken for an ordinary abscess but this is no longer a common error.

The treatment consists essentially in the removal or destruction of the tissues that make up the sinus or cyst, particularly the epithelial elements, but there is some difference of opinion as to how this is most satisfactorily accomplished. There are two general methods, one is a direct attack on the sinus itself, the other attempts to encompass and remove the tissues which contain the sinus. Either method is effective if properly applied, but recurrence or persistence has been and continues to be a problem, and the time required for healing is often long.

Direct attack upon the sinus is usually made with a destructive chemical or the cautery. Anderson,[3] who reported the first case of pilonidal sinus in 1847, opened the mouth of the sinus and used injections of silver nitrate and chloride of mercury without any perceptible benefit. He then made an opening in the cavity and three weeks later found the patient entirely well. The chemical treatment of pilonidal sinuses has not been widely adopted since this first trial, but with improved solutions and better technic more favorable results have been obtained, and the method has gained a certain number of adherents.[23, 24, 25] Rogers,[21, 26, 27, 28] and his associates, have been consistent advocates of cautery excision and this method, in their hands, has given very good results, particularly with respect to the diminution of recurrences.

The majority of surgeons, however, treat pilonidal sinuses by excision with varying amounts of the surrounding tissue, and differ mainly in the method of closing the wound. Primary closure, open packing, and a number of modifications of these basic methods have been used but few are satisfied, either with the rate of recurrence or with the healing time. The principal drawbacks to primary closure are the constant presence of infection and the difficulty in obliterating the dead space. To overcome these disadvantages Ferguson and Mecray[29] defer operation until infection is controlled, and after the excision effect a careful closure which includes the use of retention sutures of alloy steel wire. The patient is then placed on his back on a litter or operating table for an hour to insure obliteration of any residual dead space. Gage[30] believes that operation should be deferred till six months after infection. He uses fine silk ligatures, does a primary closure, and applies a sea sponge under pressure to maintain the obliteration of the cavity. In his experience, silk ligatures have given better results

than catgut. Dunphy,[31] also, has advocated the use of silk and believes it reduces the number of recurrences. Colp[32] described a method of primary closure in which the flaps are mobilized, by undermining if necessary, and then sutured separately to the midline of the posterior sacrococcygeal ligament by interrupted mattress sutures of silk. The skin edges are approximated with fine silk.

Primarily concerned with obliteration of the dead space, Lahey,[33] in 1929, introduced an operation in which a flap with a single pedicle is raised from the adjacent skin and subcutaneous tissue and moved over into the

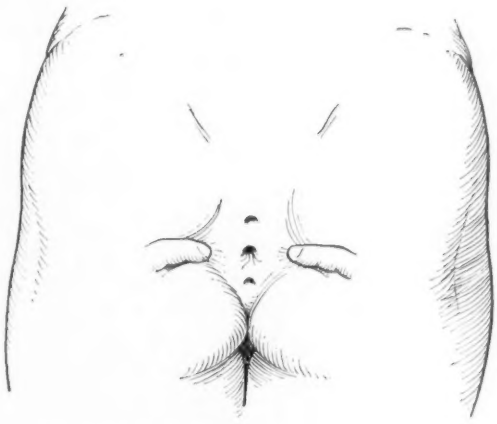

FIG. 1.—Typical appearance of pilonidal sinus, with hair projecting from middle sinus.

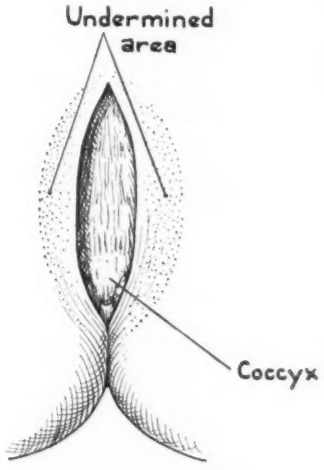

FIG. 2.—Sinus removed by block dissection, with undermining of skin and exposure of sacrococcygeal fascia.

wound. In 1932, Lahey[34] suggested that a flap be supported by two pedicles, one above and one below, instead of the original single pedicle above. Swinton and Hodge[35] state that the modified operation is occasionally used at the Lahey Clinic, but limited block-excision with light packing is the common practice.

The advantages of a successful primary closure are obvious—the healing time and the period of disability are greatly reduced. A few surgeons have reported excellent results with primary closure but in most hands the method is disappointing. The wound frequently fails to heal *per primam,* and often there is persistent drainage or recurrence.

It is probable that the majority of surgeons in this country treat pilonidal sinuses on the principle of excision with open packing of the wound. Believing that recurrences are fewer, they are willing to accept the prolonged healing time. In a collected series, Kleckner[36] reported 4,699 cases, of which 4,231 had been operated upon by the open method.

The results achieved with the standard methods of treatment show considerable divergence, as reported from various clinics and by individuals. Gage,[30] for example, records 42 cases of excision, with primary closure,

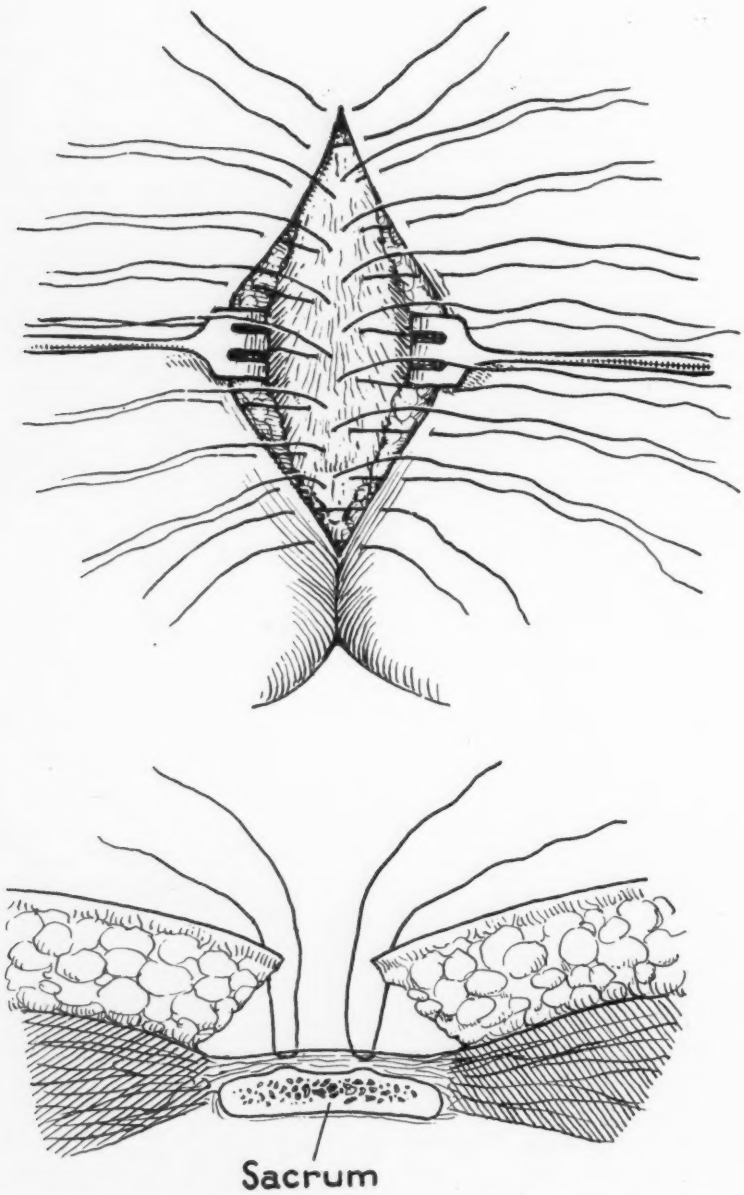

FIG. 3.—Sutures of black silk placed through skin edges and fascia ready for tying, viewed from above and in cross-section. In passing sutures through skin the subcutaneous fat is not included.

without a single recurrence, whereas in the cases reported by Rogers and Hall[27] the recurrence rate for primary closure was 36 per cent in the first series and 38 per cent in the second.

In attempting to compare the results of primary closure with the open method the same difficulty is encountered. In the large series collected by Kleckner[36] the incidence of recurrence was reported as 23.29 per

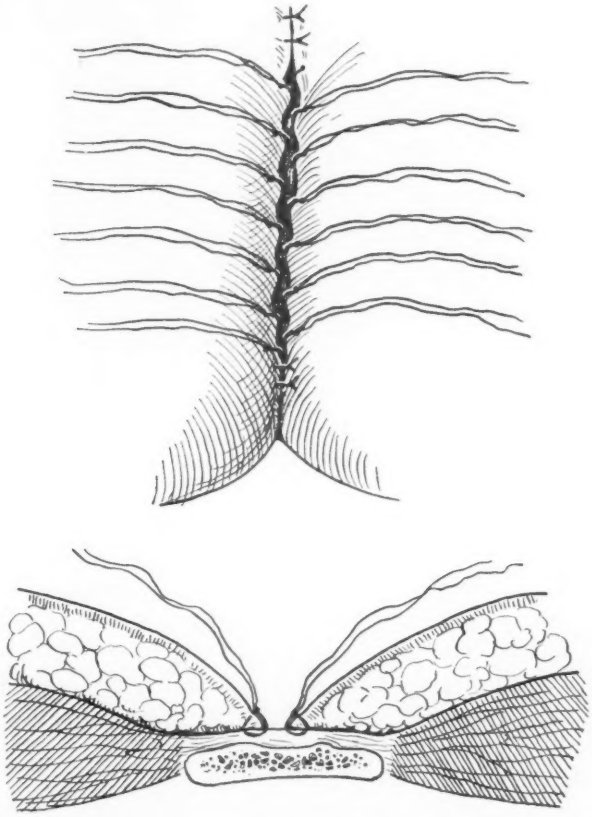

FIG. 4.—Sutures tied, bringing skin edges to fascia near midline without tension.

cent for primary suture and only 1.13 per cent for the open method. Rogers and Hall[27] found recurrence rates of 18 and 25 per cent of the cases treated by open operation in their first and second series respectively. In Kooistra's[15] series the recurrence rates for primary closure and open packing were the same—21 per cent.

The time required for healing of the operative wound is a matter concerning which complete data is not available. The advocates of primary closure have emphasized the time factor when comparing this method with open packing, but usually do not take into account the cases that fail to heal by primary union. Rogers and Hall[27] reported an average healing time of 2.7 months, approximately 82 days, following cautery excision. In the series of 29 cases treated by excision and light packing, Swinton

and Hodge[35] found that 21 cases required 16 weeks, or 112 days to heal and the remainder up to one year. In Kooistra's cases, 24 treated by excision with packing required an average healing time of 90 days after leaving the hospital; in an equal number treated by primary closure the healing period was only 24 days. Two cases that were closed primarily but later opened and packed required 94 days to heal. Rogers[21] has properly stressed the importance of the after-care of these cases, and believes it is important that the patient should remain under the treatment of the surgeon who has operated upon him.

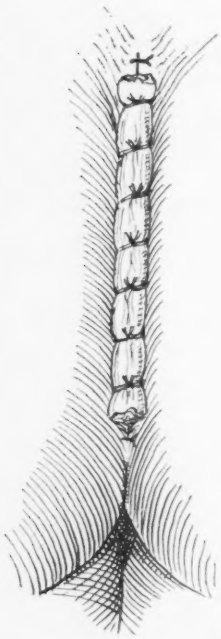

FIG. 5.—The ends of the sutures which have been left long are tied over a dressing-roll of wet cotton or gauze as a convenient means of holding it in place. The wet dressing provides a capillary medium for the absorption of serious exudate.

The type of wound closure about to be described (Figs. 1–5) has as its principal purpose the elimination of as much of the open wound as possible without the creation of a dead space. The tissue involved by the sinus is removed in the usual manner by block dissection, and partial wound closure is effected by suturing the skin edges to the sacrococcygeal fascia and underlying ligamentous structures. The skin edge on each side is brought as near the midline as possible without tension, and sutured with interrupted medium black silk, leaving a narrow uncovered area of fascia between. If this area is large enough to warrant it, a skin graft may be applied to further shorten the period of healing. A roll of gauze or cotton wet with normal saline solution is then applied to the wound, where it may be conveniently anchored by means of the suture ends which are left long to facilitate their subsequent identification and removal. If the wound can be kept clean it need not be disturbed for several days, but the dressing may be changed frequently if necessary. The sutures should not be removed until the skin has become firmly fixed to the fascia. This is usually a matter of 10 or 12 days.

Since January 1, 1936, 214 ward patients have been operated upon in New York Hospital for pilonidal sinus, with a total of 230 operations performed. The procedure described above was introduced at this hospital in 1936 and has since been carried out in 144 patients a total of 147 times, three of the patients having been operated upon twice. During the same period 36 operations with open packing, 34 with primary suture, and 12 incisions for the drainage of abscesses were performed. One other patient had a total of seven operations of various kinds and extent, and was not considered suitable for classification. With few exceptions the operations were performed by the resident staff. The treatment following discharge from the hospital was conducted in the Out-Patient Department with no particular group assigned to these cases.

The results of the various procedures are shown in Table I and are based upon operations performed rather than the number of patients operated upon. When a patient entered the hospital with a recurrence he was considered another case regardless of where the previous operation had been done. In reviewing the table, "cases" should be thought of as operations for pilonidal sinus rather than the number of patients involved. All cases including the simple incisions for drainage and the one not classified appear in the table.

The healing time is based upon the number of cases in which it could be definitely determined, and includes the days spent in the hospital after operation. Patients who had small granulating areas or slight discharge when last seen were not considered as healed. The numbers of cases upon which healing time is based are entered in parentheses.

TABLE I

THE RESULTS OF SURGICAL TREATMENT IN 230 CASES OF PILONIDAL SINUS

Type of Wound Closure	No. of Cases	Average Postop. Days in Hospital	Average Healing Time, in Days	Cured		Persistence or Recurrence		Result Unknown or Incomplete	
				No.	Per Cent	No.	Per Cent	No.	Per Cent
Suture of skin edges to sacrococcygeal fascia..	147	14	69, (89 cases)	97	66.	19	12.9	31	21.1
Packing of open wound..	36	25	118, (26 cases)	26	72.2	6	16.7	4	11.1
Primary suture.....	34	14	70, (23 cases)	22	64.7	11	32.3	1	2.9
Incision and drainage...	12	7	153, (3 cases)	6	50.	2	16.7	4	33.3
Unclassified.....	1					1			

The results with respect to cure and recurrence are based upon examinations in the Follow-Up Clinic. In a few instances information from sources considered reliable was accepted. No case was recorded as cured until followed for at least six months, the average time being 16 months. The term "cured" as applied to cases of incision and drainage should be interpreted as meaning free of symptoms for six months or more after operation. Patients who had a follow-up of less than six months were considered too recent for classification except in instances of recurrence. Percentages are based upon the total number of cases treated by each method as they appear in the first numerical column.

The average healing time following suture of skin edges to sacrococcygeal fascia was 69 days, for open packing it was 118 days, and for primary closure 70 days. Eighteen, or 53 per cent, of the 34 primary closures developed abscesses or other complications which greatly prolonged the healing time and raised the general average.

The patterns of wound healing in the cases with known healing time are shown in Charts 1, 2, and 3. The healing time is represented by the ordinates and the number of patients by the abscissas. In Chart 1 it will be observed that 47, approximately 52 per cent, of the 89 cases treated by suture of skin to fascia had healed within a period of 50 days, and 62, or 70 per cent, within 70 days. Chart 2, representing 26 cases treated by open packing of the wound, shows only 2 cases, approximately 8 per cent, healed within 50 days, and 9, approximately 35 per cent, healed within 70 days.

Chart 3, representing 23 cases treated by primary suture, shows that 14, approximately 61 per cent, had healed within 50 days, and 15, or 65 per cent, within 70 days. The numbers and percentages of cases requiring more than 150 days to heal were 6, or 6.7 per cent, for suture of skin to fascia; 6, or 23 per cent, for open packing; and 4, or 17 per cent, for primary closure. The figures dealt with are obviously too small to serve as a basis for conclusions but by providing an indication of healing tendencies in the three types of wound treatment they perhaps have a limited statistical value.

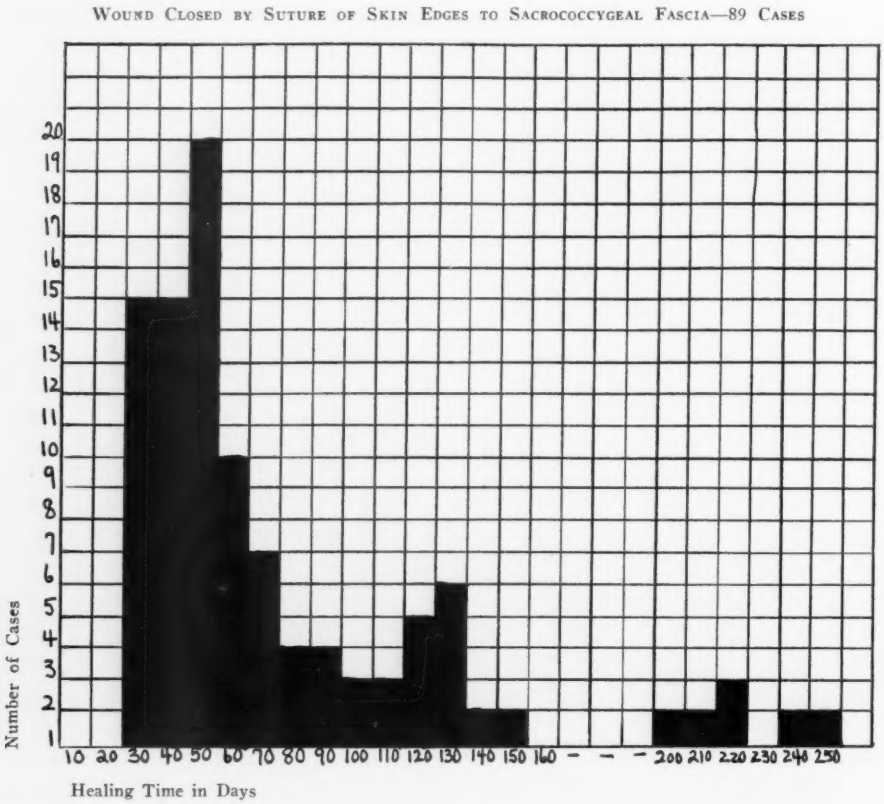

Chart 1.—Healing pattern of wounds closed by suture of skin edges to sacrococcygeal fascia. The peak of healing was reached at 50 days, and the period in which the majority of cases healed was from 30 to 70 days.

With respect to ultimate results, there were 19 known instances of recurrence or persistent drainage, 12.9 per cent, in the 147 cases of closure by suture of skin to fascia; 6, or 16.7 per cent, in 36 cases treated by open packing; and 11, or 32.3 per cent, in 34 cases closed by primary suture.

In this series the results of closure by suture of skin to fascia compare favorably in all respects with those of open packing and primary suture. The healing time following this operation is shorter than that generally recorded for the open operation. It is longer than the healing time for primary suture when primary suture is successful, but is shorter than that required

by the wound which has been closed by primary suture but subsequently opens and requires packing.[15]

Recurrence or persistence of the disease following closure by suture of skin to fascia is less frequent than that reported by Dunphy,[31] and Kooistra[15]

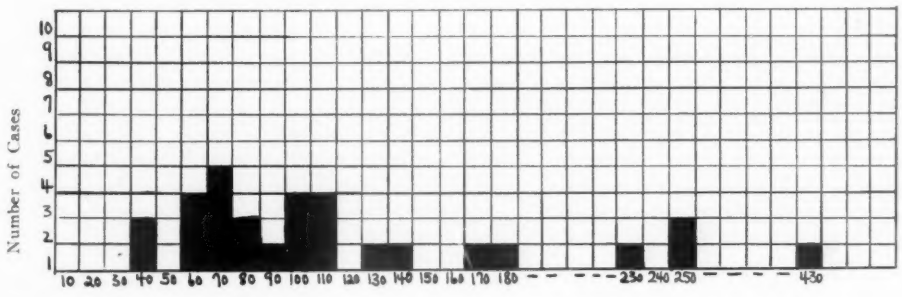

Chart 2.—The healing of wounds treated by open packing. The high point of healing was reached at 70 days. The period in which the majority of cases healed was from 60 to 110 days.

for primary closure and for open packing. It is much higher than Ferguson,[29] and Gage[30] report for primary closure, and higher than Rogers and Dwight[28] give for cautery excision with open packing. Rogers[21] is convinced that many so-called recurrences are simply infected dead spaces due to faulty healing, and one must agree with him as to the importance of proper attention to the healing wound.

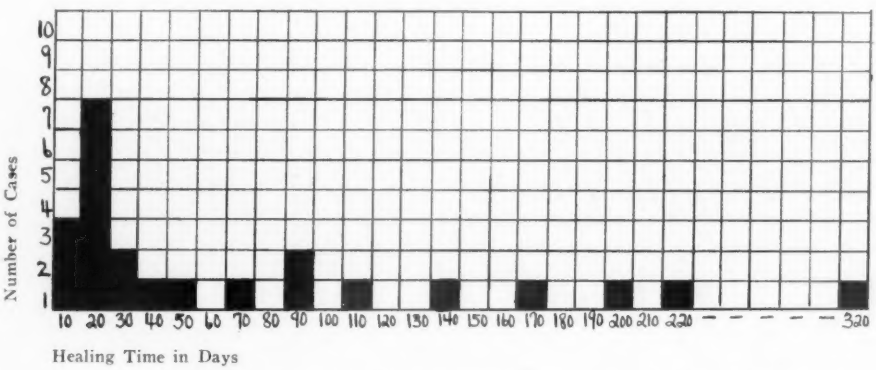

Chart 3.—The healing of wounds closed by primary suture. The high point in healing was at 20 days, and slightly more than half the cases had healed within 30 days. The remainder failed to heal *per primam* and required varying lengths of time up to 320 days.

SUMMARY

1. The series consists of 214 patients upon whom 230 operations for pilonidal sinus were performed.

2. No two cases came from the same family.

3. There was no tendency toward association with other anomalies, but an incomplete spina bifida was present in one case.

4. The ratio of males to females was three to one.

5. The series contains two Negroes, but no members of the yellow and brown races. Italians, Central Europeans, and Jews were the largest racial groups in a very mixed population.

6. All cases presented evidence of infection, and half of them gave a history of one or more abscesses which had either been incised or had ruptured spontaneously.

7. Thirty-two, or 15 per cent, had had one or more previous operations designed to bring about a cure.

8. Only 13, or 6 per cent, of the 214 patients attributed the onset of symptoms to trauma.

9. Five of the 57 female patients, 8.8 per cent, gave childbirth as the cause of onset, and two others stated that symptoms began during pregnancy.

10. The youngest patient was 15 years of age and the oldest was 55. The average age of onset was 22.5 years, and the average age at the time of admission was 25 years. Four patients stated that the sinus had been observed at birth.

11. A method of partial closure intended to reduce the healing time is presented and the results are compared with other methods of closure.

12. The initial results are encouraging but the ultimate value of the method depends upon further experience with it.

13. The postoperative care is important, both with respect to healing time and to the incidence of recurrence.

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SUCCESSFUL CLOSURE OF AN ARTERIOVENOUS ANEURYSM INVOLVING THE LEFT INNOMINATE VEIN AND THE LEFT COMMON CAROTID ARTERY

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ARTERIOVENOUS ANEURYSMS are fairly common and rather easily recognized by the classic physical signs which are invariably present, with possible minor variations. We are presenting our experiences with an arteriovenous aneurysm which was unusual in its location. The difficulties encountered in the surgical correction of the condition were considerable. Our findings are analysed and a brief summary of the syndrome is presented.

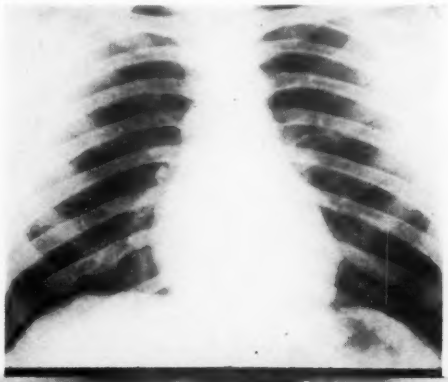
Case Report.—B. J., Negro, male, age 31, was admitted to Grasslands Hospital, June 26, 1939. *Chief Complaint:* Unilateral swelling and pain of left side of face of four months duration. *Past History:* Essentially negative. Malaria as a child. History of luetic infection in 1934, with subsequent treatment. *Present Illness:* Began January 17, 1939, when patient was stabbed in the chest. The White Plains Hospital reported a half-inch laceration of the skin, located about one-half inch below the sternal notch over the manubrium sterni, to the left of the midline, which was surrounded by a hematona. The patient expectorated grayish blood-streaked sputum. The laceration was sutured and drained. The patient remained in the White Plains Hospital for four days, during which time roentgenograms of his chest were taken. He left the hospital against advice.

In February, 1939, about one month after the accident, the patient began to notice swelling of the left side of his face. Two weeks later he noted a buzzing in his left ear on turning his head to the left. He gradually developed shortness of breath which, on admission, had increased to the point where dyspnea was present on climbing two short flights of stairs. Shortness of breath was also initiated by lying on the left side. During the three months prior to admission, he had a cough productive of three to four ounces of thick sputum per day. The swelling of the left side of the face gradually became more marked and finally involved the left side of the neck. This swelling receded during the daytime, only to return during the night while the patient was lying down. During the weeks previous to his admission the buzzing in his left ear had become more annoying and was present most of the time. For three months previous to admission he had noted soreness under the upper sternum, aching pains down the left arm, and pain in the temporal region.

Physical Examination.—Positive Findings: On inspection, there was an obvious unilateral swelling of the left side of the face involving the left cheek, left lower eyelid, the temporal and parietal regions, and extended down over the mandibular region, and entire left side of the neck. The swelling was soft and did not pit. There was no exophthalmos; the pupils were equal and reacted normally; fundi were normal. On auscultation, a double bruit could be heard over the greater part of the swelling, but was loudest over the carotid region of the left side of the neck. The veins over the entire left side of the face, head and neck were swollen and distended. A palpable systolic thrill was present over both carotids, more marked on the left.

Examination of the chest showed an old, healed scar of the short, diagonal, three-quarter-inch stab wound over the manubrium sterni, slightly to the left of the midline, and about one inch below the sternal notch. The veins over the left side of the chest, left shoulder region, and entire left arm, as well as over the left upper portion of the abdomen were distended. A thrill, systolic in time, was felt over the entire cardiac area, but was most marked over the upper anterior chest. There was no pulsation in the sternal notch, no brassy cough, nor tracheal tug.

FIG. 1.



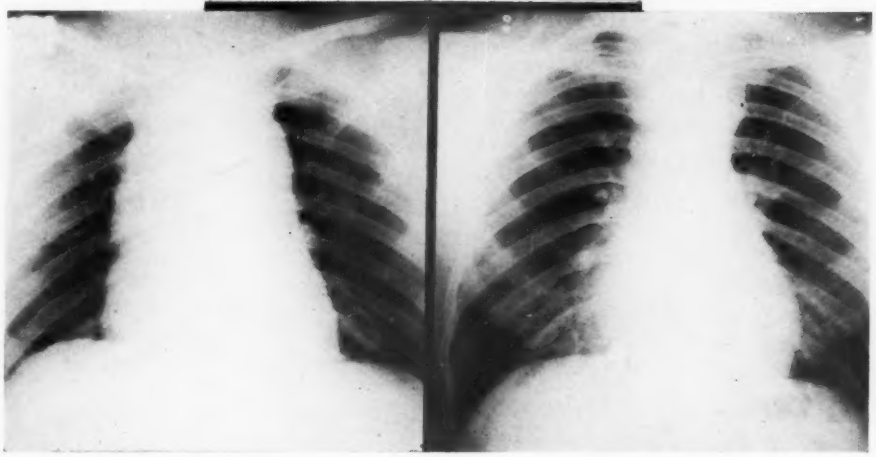


FIG. 2. FIG. 3.

FIG. 1.—Six-foot roentgenogram of the heart, September 24, 1936, two and one-half years before the stabbing showing a normal heart and clear lung fields.

FIG. 2.—Roentgenogram of chest taken immediately following the accident, patient in prone position, showing a marked widening of the mediastinum, especially superiorly.

FIG. 3.—Six-foot roentgenogram taken in July, six months after the injury, showing a widening of the superior mediastinum and generalized cardiac enlargement.

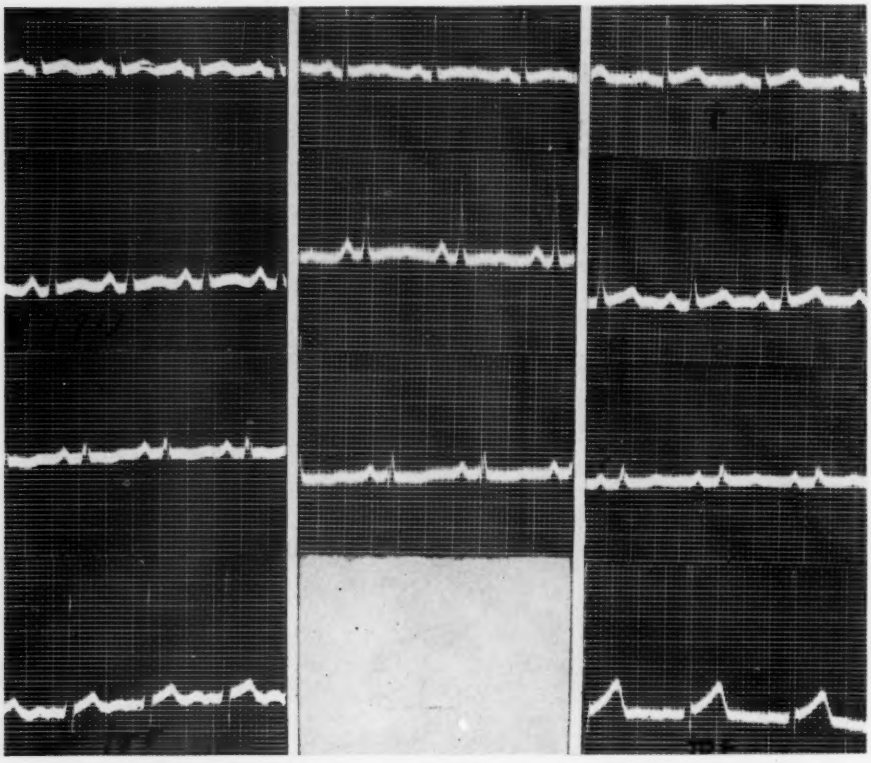
The heart was enlarged in the transverse diameter. A loud, continuous murmur was heard over the entire precordium, which was loudest in the region of the scar. The quality and transmission of the diastolic murmur was very similar to that found in aortic insufficiency. The pulse was Corrigan in quality and there was a "pistol-shot" sound audible over the femoral vessels. Blood pressure 140/60; equal on both sides.

The left upper extremity, including forearm and hand was swollen, as compared to the right. The left arm measured 28½ cm. in circumference, 12 cm. above the elbow, and 32½ cm., 12 cm. below the elbow. The right arm measured 28 cm. in circumference, 12 cm. above the elbow, and 32 cm., 12 cm. below the elbow. Pulse

88–100, respirations 18–22, at rest. The remainder of the physical examination was essentially negative.

Laboratory Data.—Venous pressure—left 35 cm. (350 Mm.) water; right 14 cm. (140 Mm.) water. Circulation time (calcium gluconate)—left 19 seconds; right 16 seconds. Wassermann and Kline tests negative. Urine negative. Hb. 14.5 Gm. R.B.C. 4,550,000. W.B.C. 4,700, differential count 52% neutrophils, 46% lymphocytes, 2% monocytes. Blood sugar 80 mg. N.P.N. 25 mg. CO_2 combining power—left arm 63; right arm 63.

There had been occasion, in 1936, for a chest roentgenogram, which we were fortunate enough to obtain for comparison with the films made subsequent to the stabbing injury:



GRAPH. 1.—Tracing A was taken two weeks preoperative. Tracings B and C were taken 11 days, and three months postoperative. The essential difference is in the height of the T-waves. Those in tracing C are much higher than those of either A or B.

Roentgenologic Examinations.—(1) The 1936 films showed the mediastinum of normal width, the heart was not enlarged, and there was no evidence of pleural fluid (Fig. 1).

(2) The films taken January 17, 1939, directly after the accident, showed marked widening of the mediastinal shadow, particularly in the upper portion (Fig. 2).

(3) Films taken in July, six months after the injury, showed a widening of the superior mediastinum, and generalized enlargement of the heart (Fig. 3).

An electrocardiogram, July 5, 1939, was within normal limits (Graph 1).

This case was investigated by the medical department and the diagnosis of arteriovenous aneurysm between the left innominate vein and the left common carotid artery

was arrived at. A surgical consultation was requested and the decision to operate was based on the following considerations:

First, for the relief of tinnitus, pain and swelling of the face and neck. Second, but most important, for the alleviation of the altered dynamics which had caused cardiac enlargement and disability that would undoubtedly have been progressive. It was felt that a sufficient length of time had elapsed to allow collateral circulation to be established, in case ligation was found to be necessary.

Operation.—July 18, 1939: Under nitrous oxide, ether and oxygen anesthesia, with a closed system and endothracheal tube, a vertical midline incision was made from the midpoint (hyoid bone) on the neck, downward to the junction of the upper and middle thirds of the sternum. The sternum was divided in the midline from the sternal notch to the level of the third interspace. The sternal incision was carried to the left just below the third rib. The upper left sternal segment still attached to left clavicle, first, second, and third left ribs, was then elevated. The pleura was separated bluntly from the deep surface of the sternum and ribs. A mass completely filling the area, exposed by elevating that portion of the bony framework, obscured the structures beneath. Investigation of this mass, which was soft and pulsating, showed it to be made up of very friable tissue. It was ovoid in shape, and measured about seven centimeters in width and ten centimeters in length. By careful dissection below this mass the arch of the aorta was identified. The innominate vein, as such, could not be accurately demonstrated except at the distal end of the mass, where a structure resembling a dilated large vein coursed outwards beneath the clavicle. The internal jugular vein was identified in the lower neck close to the common carotid, and this vein was likewise dilated more than normal. It was concluded that this mass represented a pulsating aneurysm of an arteriovenous fistula between the left innominate vein and probably the left common carotid artery. In attemping to encircle the distal portion of the mass by blunt dissection for purposes of ligation, its wall was torn. Hemorrhage could not be controlled by suture-ligature because of the friable nature of the sac. A curved Kelly clamp, carefully applied over the rent, stopped the bleeding. A further attempt to separate the midportion of the sac from the aortic arch, in the hopes of applying a ligature in this location, resulted in a second tear which was controlled by a straight Kocher hemostat. At this point, digital pressure was applied in an attempt to find out if the communication between the artery and the sac could be obliterated. That portion of the sac overlying the proximal end of the carotid artery was squeezed between the fingers, and immediately the pulsation and thrill disappeared from the sac. The anesthetist observed that with the application of this pressure, the thrill in the upper neck abruptly ceased. It was then felt that if a ligature could be applied surrounding this portion of the sac, in all probability, the fistula would be obliterated. In spite of proceeding with extreme caution, to dissect a space to pass a ligature, a third opening was made into the sac, with rather brisk hemorrhage resulting. A straight Kocher clamp controlled this bleeding. It now became evident that it would be impossible to ligate, suture or transfix any portion of the mass. However, with the three clamps in place, hemorrhage was controlled and the fistula was apparently closed. Consequently, the clamps were left *in situ*, and iodoform gauze was packed around them. Dr. Rudolph Matas[1] states that: "The use of hemostatic clamps allowed to remain *in situ* have saved many lives, or at least bridged over many critical situations, when ligations were impracticable. I know this is true in my own experience." The elevated left upper chest wall was approximated to its other half, the clamps coming out through the opening in the sternum. The soft tissues were approximated, and the clamps bound securely together, and a suitable dressing applied.

Postoperatively, the patient had a fairly uneventful course. He developed a mild infection in the wound, probably because of the packing and clamps. On the twelfth day postoperative two of the clamps were carefully removed, without mishap. The third clamp was removed on the fourteenth postoperative day, with no untoward results.

The packing was removed bit-by-bit, and the infection treated.

A roentgenogram, August 16, 1939, 30 days postoperative, showed a reduction in the size of the mediastinal shadow, as well as a reduction in the size of the heart to normal (Fig. 4).

An electrocardiogram, July 29, 1939, 11 days postoperative, showed flattening of the T-waves (Graph 1). By September 22, 1939, 65 days postoperative, the T-waves had increased in amplitude to a point where they were higher than they were on July 5, 1939 (Graph 1).

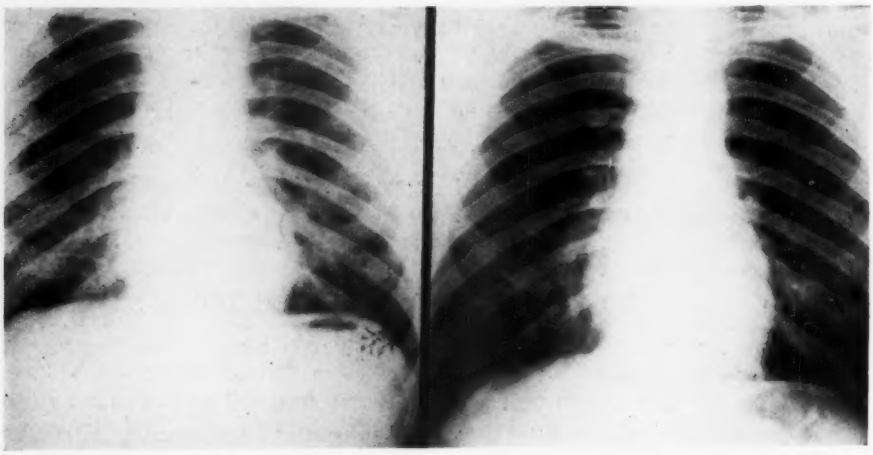


FIG. 4.—Six-foot roentgenogram, August 16, 1939, one month postoperatively showing narrowing of the superior mediastinum, and a reduction in the size of heart towards normal.

FIG. 5.—Six-foot roentgenogram, October 7, 1939, three months postoperative showing further narrowing of the superior mediastinum and a normal cardiac shadow.

The patient continued to show improvement. Some four weeks after operation a rather profuse, sudden hemorrhage occurred from the depths of the wound, but this was fairly easily controlled by packing. Several minor episodes of bleeding occurred after this, and a low-grade infection persisted, but the wound slowly granulated. A small abscess developed over the left upper chest, some seven centimeters from the main wound, which was incised and drained. Cultures from this abscess showed *Staphylococcus albus*.

On September 24, 1939, 67 days after operation, the patient left the hospital against advice. At this time the patient offered no subjective complaints. He had no pain, or respiratory distress. He felt no buzzing in his ear or head. The swelling of the left face and head had completely disappeared. There was no evident swelling of the left shoulder or left arm, though a slight fullness persisted in the left supraclavicular region. The distended neck veins had receded to normal size. The previously heard loud murmurs and cardiac thrill had disappeared. A low systolic blowing murmur was heard at the apex, which was transmitted to the left axilla. No bruit could be heard in the chest or in the neck. The heart rate had slowed to 80, and less. The blood pressure was approximately equal on both arms (left 98/70, right 100/74). Venous pressures—right 12.5 cm. (125 Mm.) water, and left, 30.5 cm. (305 Mm.) water.

The patient was seen July 26, 1941, at which time he was working in a munitions factory lifting 75 to 100 lbs. This caused him no discomfort. Blood pressure 126/76. Pulse 78. No thrills or murmurs. Fluoroscopy and electrocardiogram normal.

DISCUSSION.—Arteriovenous fistulae may be either acquired or congenital. Both types produce local circulatory disturbances, and, if large enough,

profound systemic effects. The most common arteriovenous aneurysms are those involving the femoral, axillary, brachial, popliteal, carotid and subclavian vessels.[4] Our case is unusual, in that there is only one other recorded in which a fistula has connected the left internal carotid artery and the left innominate vein.[14] The other instance was overlooked during life, and was the ultimate cause of death.

The largest arteriovenous aneurysms are those connecting the aorta and the superior vena cava.[2] They are practically always luetic in origin, and are invariably fatal within a short time. Recently, Burwell[3] introduced the interesting conception of considering the placental circulation as an arteriovenous aneurysm. His points are well made and quite fascinating to contemplate. The most common congenital arteriovenous aneurysm is the patent ductus arteriosus.

The classic local signs of a continuous bruit and thrill, which are accentuated during systole and transmitted along the line of the vessels may be modified by the position of the arteriovenous aneurysm. In our case a diastolic thrill could not be felt, probably because of the chest thickness. The history of lues, the increased pulse pressure, Corrigan pulse, "pistol-shot" sound over the femoral arteries, and the character and transmission of the diastolic murmur made it necessary, for a while, to consider the possibility of a concomitant aortic valve lesion.

The local manifestations, in most instances, are of little consequence; a notable exception is the arteriovenous aneurysm involving the internal carotid artery and cavernous sinus, in which condition, the local changes greatly overshadow the systemic disturbances.[21] In the case herein presented, the patient sought relief for the local swelling, pain and bruit.

The systemic phenomena which may occur with an arteriovenous aneurysm are: (1) An accelerated heart rate; (2) an elevation of the systolic arterial blood pressure and a lowering of the diastolic pressure, with a resultant increased pulse pressure; (3) an increased cardiac output, with a decreased stroke output; (4) engorgement of the pulmonary vessels; (5) an increased circulating blood volume; (6) cardiac enlargement; (7) electrocardiographic changes; and (8) increased venous pressure and circulation time, with the onset of cardiac decompensation.[3, 12, 17, 18, 19] Where the fistula is accessible, compression will cause immediate slowing of the heart rate (Branham's sign), and a lowering of the systolic arterial blood pressure. These changes may be of assistance in evaluating the degree to which the arteriovenous aneurysm is influencing the general circulation. In some instances it has been possible to witness, under fluoroscopic examination, a clearing of the pulmonary fields upon digital obliteration of the shunt.[12]

The position of the fistula in our case, precluded its compression before operation. Following surgical correction, however, the anticipated reversals were observed. The changes in heart size were the usual thing. The enlargement which occurs with arteriovenous fistulae is considered to be 75 to 90 per cent dilatation and 10 to 25 per cent hypertrophy.[7] The longer

the communication remains patent the more genuine hypertrophy develops and the less likely is complete reversibility possible.[17]

The fullness of the left supraclavicular area, and the elevated venous blood pressure in the left upper extremity which have persisted after operation in our patient are, we feel, the result of a marked narrowing of the lumen of the left innominate vein. The cardiac rate has slowed from 88–100 to 78. The systolic blood pressure which was 140 before operation fell to 100 after operation, while the diastolic blood pressure rose from 60 to 70. While others have recorded improvement in the electrocardiographic picture following obliteration of the fistula, the changes in the T-waves noted here are perhaps more marked than usual.[19]

The effects of an arteriovenous aneurysm are dependent upon the size of the fistula, the caliber of the vessels involved, the distance of the fistula from the heart, the volume and force of the arterial stream which is short-circuited, the age of the patient and his activity, and the presence or absence of coexistent cardiovascular disease.[9, 13, 15, 17] Variations and combinations of the above factors explain the differences that one finds in the description of cases in the literature.

The time-honored surgical treatment for arteriovenous aneurysms has been a four-point ligation and removal of the fistula.[8, 9, 10] The excellent results of Gross in the simple ligation of patent ductus arteriosus,[11] and our own end-result are evidence to the fact that radical procedures are not always necessary. Correction through transvenous arteriorrhapy is frequently the method of choice.[16] In desperate situations, where cardiac decompensation is severe, one may have to resort to simple ligation of the proximal portion of the vein for temporary alleviation. The fact of the matter is that it is often impossible, and occasionally extremely dangerous, to perform a four-point ligation.[20] Each instance of an arteriovenous aneurysm requires careful thought and precision in judgment as to the proper surgical procedure; the age of the patient, the duration of the aneurysm, the vessels involved, the type of fistula present, *etc.*, will all influence the type of approach necessary, and very often the decision will have to be altered at the last moment to meet the findings disclosed at operation.

Unfavorable effects upon the general circulation are the usual indication for surgical intervention. Occasionally local changes alone may be troublesome or serious enough to require correction. In patent ductus arteriosus the question has arisen as to whether one is justified in ligating the fistula solely on the possibility that bacterial endocarditis may be obviated.[11] The *Streptococcus viridans* has been known to lodge at the side of a traumatic arteriovenous aneurysm, causing all the signs and symptoms of a subacute bacterial endocarditis. Hamman and Rienhoff[6] reported the surgical cure of such a case.

SUMMARY AND CONCLUSION

(1) A case of arteriovenous aneurysm involving the left innominate vein and the left common carotid artery is reported.

(2) The local and general manifestations of arteriovenous aneurysms are listed.

(3) The method resorted to in the correction of the arteriovenous aneurysm reported in this paper, is described. This method is not advocated as an orthodox procedure but was dictated by the complications which arose.

(4) The methods and indications for the correction of arteriovenous fistulae are mentioned.

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A COMPLICATED CASE OF ANEURYSM INVOLVING THE ILIAC AND FEMORAL ARTERIES

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THE FOLLOWING CASE of iliofemoral aneurysm is reported because of several unusual features which complicated its management:

Case Report.—C. C. H. No. 145047: The patient, F. F., came under observation May 26, 1941. He gave the history that 38 years ago he had been shot through the inner aspect of his left thigh. An "aneurysm," probably an arteriovenous fistula, developed at the site of the wound and was excised. Three years later a pulsating mass in the left groin spontaneously appeared. During the past 35 years this mass had slowly increased in size and was becoming painful. In addition, he was suffering from chronic ulceration of the leg, intermittent claudication, and extreme coldness, numbness, and cyanosis of the foot.

Physical Examination.—White male, age 60. The left foot was extremely cyanotic, cold to the touch and moist. On elevating the limb the sole of the foot became pale. Atrophy and ulceration of the skin of the lower two thirds of the leg were present. Pulsation was barely palpable in the dorsalis pedis and posterior tibial arteries, and oscillometric examination revealed only a trace of pulsation in the region of the lower leg. There was a depressed linear scar six inches in length overlying the course of the subsartorial canal in the thigh, and no arterial pulsation was palpable in this region.

The common femoral and external iliac arteries had been converted into a large, painful, vigorously pulsating, fusiform aneurysmal mass. The aneurysm was constricted by the overlying ilio-inguinal ligament giving it a dumb-bell shape. The femoral component of the aneurysm measured four inches in its external diameter. Serologic tests for syphilis were negative.

To evaluate what rôle vasospasm played in the evident ischemia of the foot and leg, skin temperature readings were taken before and after a procaine block of the posterior tibial nerve. At a room temperature of 26° C., the nerve block was followed by a rapid rise in the surface temperature of the fifth toe and adjacent sole, from a stabilized level of 25° C. to a vasodilation level of 32° C., and the cyanosis of the toes was replaced by a healthy pink color (Chart 1).

Because of this obvious vasospasm, it was deemed advisable to precede excision of the aneurysm by a lumbar sympathectomy. It was felt that permanent relaxation of these spastic vessels would expedite the growth of a distal collateral circulation and constitute a protection against gangrene of the ischemic foot which, in the absence of a superficial femoral artery, might otherwise follow excision of the external iliac and common femoral arteries. Accordingly, on June 11, 1941, through a muscle-splitting extraperitoneal approach, the left lumbar sympathetic trunk was divided at the upper margin of the third lumbar vertebra, stripped for a length of four inches, and buried in the adjacent psoas muscle.[1,2] The following day, the foot and leg were hot, red, and dry. Figure 1 shows the external appearance of the femoral portion of the aneurysm several weeks following sympathectomy.

At the time of his second admission to the hospital, four months following the sympathectomy, it was noted that the foot and leg were now pink and warm and that the ulceration had healed. Oscillometric readings taken at the level of the lower leg had increased from a preoperative value of one-half to one and one-half. The aneurysm had reached alarming size.

On October 13, 1941, the femoral portion of the aneurysm was exposed through a vertical incision. Distally, the aneurysmal sac terminated in a fibrosed, cord-like structure which, on further dissection, proved to be the proximal remains of the previously resected superficial femoral artery. The deep femoral artery, which exhibited strong pulsation, emerged from the posterior aspect of the lower portion of the sac. On mobilizing the lateral margin of the sac, the femoral nerve was observed to be imbedded in the wall of the aneurysm. Resection of the sac could not have been accomplished without injury to the femoral nerve. A length of umbilical tape was passed around the sac just above the orifice of the deep femoral artery and tied.

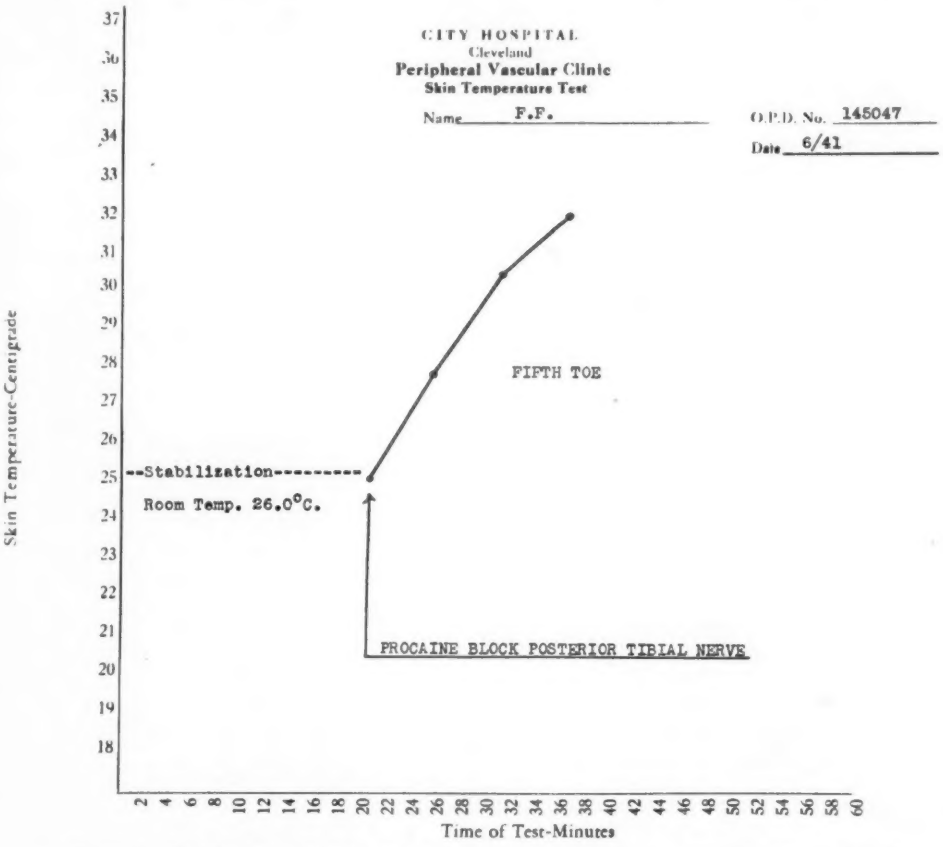

CHART 1.—Depicting the rapid rise in the skin temperature following temporary interruption of the flow of sympathetic nervous impulses to the vasospastic foot.

Pulsation in the deep femoral artery promptly disappeared, and there was a distinct diminution in the vigor of pulsation within the aneurysm itself. Nutrition of the foot was maintained.

On October 27, 1941, the iliac portion of the aneurysm was exposed through a supra-inguinal extraperitoneal approach, similar to that conventionally employed to expose the lower end of the ureter. The aneurysm extended up to the origin of the internal iliac artery. A ligature of umbilical tape was placed just distal to the origin of this artery, and the entire aneurysmal portion of the external iliac together with the accom-

panying vein were excised down to their disappearance into the thigh. (Figure 2 is a diagrammatic representation of the operative findings and procedure). Microscopic examination of the resected portion of the aneurysm revealed arteriosclerosis to be the basic pathology. Pulsation within the residual femoral portion of the aneurysm sac promptly disappeared. By the time of his discharge, the sac had completely thrombosed. The foot remained pink and warm.

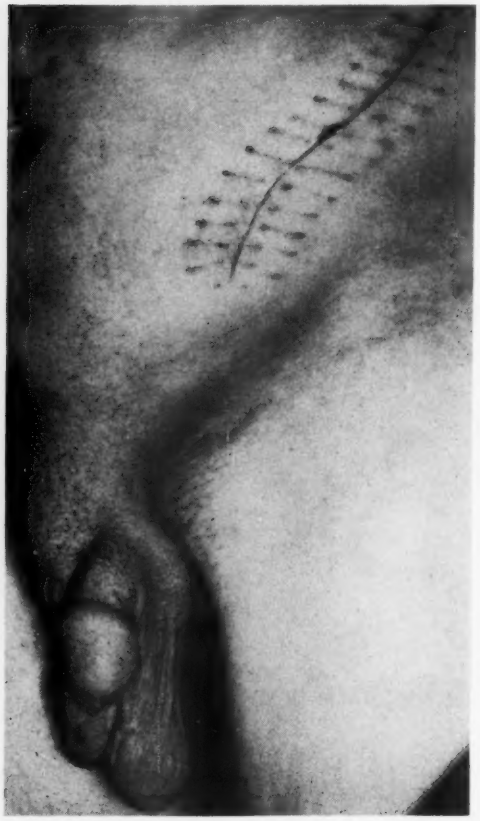

FIG. 1.—Showing the external aspect of the femoral portion of the aneurysm. Note the recently healed Pearl-type of incision employed to expose the lumbar sympathetic trunk. There is an associated right inguinal hernia.

Six months later the foot was pink and warm, and in an excellent state of nutrition. The leg ulcers had remained healed. There were no symptoms of ischemia other than a mild degree of intermittent claudication. The residual portion of the aneurysm had been converted into a fibrosing, nonpulsatile mass which had already shrunk to about one-quarter of its original size.

COMMENT.—It is the opinion of Sir Thomas Lewis,[3, 4] based on the work of Thoma,[5] and Nothnagel,[6] that the development of a collateral circulation is a true hyperthrophic response to an increase in the volume rate of blood flow through the auxilliary channels. This increase in blood flow is, in turn, a physiologic response to a diminution in the peripheral resistance to blood flow resulting from a relaxation of the arteriolar-capillary bed within the ischemic part.

Leriche,[7] and his coworkers, were able to experimentally demonstrate in dogs how sympathetic vasoconstrictor impulses inhibit the spontaneous development of a collateral circulation following resection of the bifurcation of the abdominal aorta.

Working with selected cases of arteriosclerotic disease of the lower extremities, I[8] have been able to demonstrate that there is a slowly progressive increase in the magnitude of measurable arterial pulsation in the region of the lower leg over a period of 6–12 months following lumbar sympathectomy. For reasons stated in the original communication, these observations can be explained only on the basis of a development of a collateral circulation in response to the surgically induced relaxation of the peripheral small vessel bed.

The reported case supports this conception that increased sympathetic vasoconstrictor tone may play an important rôle in the failure of a collateral circulation to spontaneously develop following a major arterial block. For 35 years this patient had suffered from severe ischemia of a foot and leg incident to excision of his superficial femoral artery. Ordinarily, in the

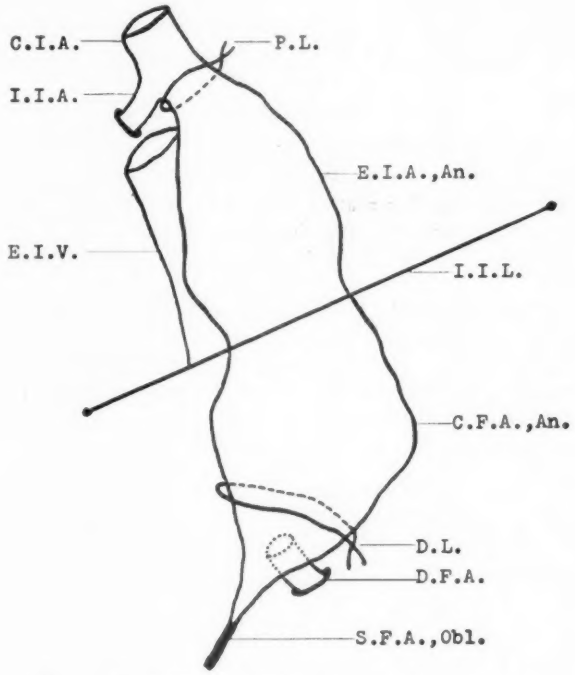

FIG. 2.—Diagrammatic sketch of operative findings and procedure.
C.I.A.: Common iliac artery.
I.I.A.: Internal iliac artery.
P.L.: Proximal ligature.
E. I. V.: External iliac vein; excised along with
E.I.A.: Aneurysmal external iliac artery.
I.I.L.: Ilio-inguinal ligament.
C.F.A.: Aneurysmal common femoral artery.
D.L.: Distal ligature.
D.F.A.: Deep femoral artery.
SF.A.: Obliterated remnant of superficial femoral artery.

absence of other peripheral arterial disease, excision of this artery does not produce such prolonged disability. The inadequacy of the collateral circulation in this instance can be best explained on the inhibiting influence of the associated vasospasm. To have thrown an added burden on this meager collateral circulation by primarily excising and thrombosing the aneurysmal iliofemoral artery would surely have resulted in gangrene of the foot and leg. For that reason it is believed that the lumbar sympathectomy was a crucial maneuver in the maintenance of an adequate blood supply to the foot and leg.

It is interesting to speculate on the anatomy of the ultimate collateral circulation present in this case. The net-result of the arterial surgery performed on this patient was equivalent to a total ablation of the main arterial

supply of a lower extremity. Beginning at the origin of the external iliac artery, it included the total lengths and all the orifices of their branches of the external iliac, the common femoral, and the superficial femoral arteries. The collateral circulation to the foot and leg must have its origin then in the buttock and posterior aspect of the thigh through anastomses between the gluteal branches of the internal iliac artery and the perforating branches of the deep femoral artery; and, finally, through communications between the latter and the geniculate branches of the popliteal artery.

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A COMPARISON OF THE RESULTS OF ROENTGEN RAYS, SULFANILAMIDE AND SERUM THERAPY IN EXPERIMENTAL GAS GANGRENE IN THE PIGEON

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THE PROBLEM of the management of wounds contaminated with the *Clostridium welchii,* and associated organisms, is not as yet settled. Numerous communications by Kelly[1, 2, 3, 4, 5, 6, 7] would lead one to believe that roentgenotherapy affords a specific with which to treat such lesions. In a review[8] of 222 cases of gas gangrene following trauma collected from the literature between 1930 and 1936, 41 cases were treated with roentgen rays and serum, with a mortality of 19 per cent; 124 were treated with serum alone, with a mortality of 17 per cent, and 57 received neither roentgen rays or serum, and had a mortality of 31 per cent. These data led us seriously to question the value of roentgenotherapy. The following experiments on pigeons were undertaken several years ago to determine whether roentgen rays would prove beneficial in experimental *Cl. welchii* infection. At the same time, the opportunity presented itself to determine the effect of sulfanilimide on similarly inoculated pigeons.

The strain of *Clostridium welchii* used in these experiments was isolated from a patient admitted to the Philadelphia General Hospital, November 2, 1937, 30 hours after a gunshot injury of the left forearm, which was self-inflicted. On admission, the clinical diagnosis of gas gangrene was made and later confirmed by bacteriologic study. The use of this strain minimized the possibility of strain specificity to which one might attribute the results obtained from use of serum if a laboratory strain had been used. The patient recovered following the use of serum and roentgenotherapy, and delayed amputation. The conscientious surgeon naturally hesitates to withhold any agent which may be of benefit to the patient. The multiplicity of therapeutic methods which are frequently employed on patients with gas gangrene makes it difficult to obtain satisfactory data as to the specificity of any one agent.

Technic.—A chopped meat proteose-peptone broth, with 5 per cent dextrose, was inoculated from the stock culture and incubated under anaerobic conditions for 16 hours. A subculture from this was made and incubated for five hours. Five cubic centimeters of the culture were then taken and centrifuged for 20 minutes. The supernatant fluid, containing most of the toxin, was removed. The bacteria were resuspended in 15 cc. of normal saline and again centrifuged. This was repeated once. Finally, the bacteria were again resuspended in 15 cc. of saline, thus having a washed 1:3 dilution of a five-hour culture. In this way virulent organisms were obtained

with a minimum amount of toxin present. The breast of each pigeon used in these experiments was injected with 0.5 cc. of this bacterial suspension.

Bull and Pritchett[9] had recommended the use of the pigeon, and we found it to be an excellent laboratory animal. All experiments were carried out in groups of 25 pigeons. These were subdivided into groups of five, so that in each experiment five groups, treated by different methods and inoculated from the same subculture were compared. In all experiments one group was used as a control. The pigeons varied in weight from 7 to 16 ounces. There seemed to be no relation between weight of pigeon and chance of recovery. The average weight of the ones that died was 11.1 ounces, while in those that lived it was 10.7 ounces. The smallest pigeon, weighing seven ounces, recovered following the use of serum.

RESULTS

Table I gives the results obtained in each experiment. They are consistent throughout and show only slight variations between groups. Table II is simply a summary of Table I.

Control.—In the control group of 30 pigeons, 63 per cent were dead within 10 to 12 hours. Only two birds survived one week, at which time the experiment was terminated. It was noted in this group, as in all others, that the birds tended to die within 24 hours and if they survived 24 hours, permanent survival was a likely possibility.

Serum-Treated Group.—The serum used in these experiments was unconcentrated horse serum and contained 300 units to one cubic centimeter. In each instance 0.5 cubic centimeter was injected into the left breast of the pigeon. This dosage of 150 units would compare roughly by weight to 40,000 units in a 150-pound man. The time of injection of the serum varied from 35 to 142 minutes after inoculating the pigeon with organisms. The average time was 83 minutes.

There was some variation in average time elapsing between inoculation and injection of the serum in the six experiments. In Experiment 1, the average time was 75 minutes; Experiment 2, 72 minutes; Experiment 3, 38 minutes; Experiment 4, 137 minutes; Experiment 5, 110 minutes; and Experiment 6, 66 minutes. Mortality was not increased by delaying the injection of serum within these time limits.

In the pigeon, serum was by far the best therapeutic agent. Only one of the 30 pigeons died within 10 to 12 hours, while 19 of 30 in the control group died in this time. Nineteen pigeons, or 64 per cent of the serum-treated pigeons, survived. It is possible that a still higher percentage of survivals could have been obtained had the injection of serum been repeated in 24 hours. That this is a specific effect, and not due to a non-specific serum reaction, is shown by experiments with normal horse serum and bovine pneumococcic serum. With the bovine serum the final mortality was 100 per cent. In Experiment 5, normal horse serum appeared to give some protection.

Sulfanilamide-Treated Group.—Fifteen pigeons received sulfanilamide by mouth following inoculation. The sulfanilamide was mixed with 5 per cent gum acacia, so that each cubic centimeter of mixture contained 100 milligrams of sulfanilamide. Each pigeon in the group received 100 mg. immediately after injection, while in Experiments 2 and 3 another 100 mg.

TABLE I

Treatment and Experiment No.	10-12 Hours			24 Hours			48 Hours			1 Week		
	Alive	Dead	Mortality %	Alive	Dead	Mortality %	Alive	Dead	Mortality %	Alive	Dead	Mortality %
Control												
1	2	3	60	0	5	100	0	5	100	0	5	100
2	2	3	60	1	5	80	1	4	80	1	4	80
3	3	2	40	1	4	80	0	5	100	0	5	100
4	1	4	80	0	5	100	0	5	100	0	5	100
5	1	4	80	1	4	80	1	4	80	0	5	100
6	2	3	60	1	4	80	1	4	80	1	4	80
	11	19	63	4	26	86	3	27	90	2	28	93
Serum												
1	5	0	0	3	2	40	2	3	60	2	3	60
2	5	0	0	4	1	20	3	2	40	2	3	60
3	5	0	0	5	0	0	4	1	20	4	1	20
4	5	0	0	5	0	0	5	0	0	5	0	0
5	5	0	0	5	0	0	4	1	20	4	1	20
6	4	1	20	4	1	20	2	3	60	2	3	60
	29	1	3	26	4	13	20	10	33	19	11	36
Sulfanilamide												
1	2	3	60	2	3	60	1	4	80	0	5	100
2	4	1	20	2	3	60	1	4	80	0	5	100
3	1	4	80	0	5	100	0	5	100	0	5	100
	7	8	53	4	11	74	2	13	86	0	15	100
X-ray, prophylactic												
1	5	0	0	1	4	80	0	5	100	0	5	100
2	1	4	80	0	5	100	0	5	100	0	5	100
3	3	2	40	2	3	60	1	4	80	1	4	80
4	0	5	100	0	5	100	0	5	100	0	5	100
	9	11	55	3	17	85	1	19	95	1	19	95
X-ray therapy												
1	3	2	40	0	5	100	0	5	100	0	5	100
2	3	2	40	2	3	60	2	3	60	1	4	80
3	2	3	60	0	5	100	0	5	100	0	5	100
4	1	4	80	1	4	80	1	4	80	1	4	80
	9	11	55	3	17	85	3	17	85	2	18	90
X-ray and serum												
4	3	2	40	3	2	40	3	2	40	3	2	40
5	5	0	0	5	0	0	4	1	20	3	2	40
6	4	1	20	3	2	40	1	4	80	1	4	80
	12	3	20	11	4	26	8	7	46	7	8	53
Normal horse serum												
5	3	2	40	3	2	40	3	2	40	3	2	40
6	0	5	100	0	5	100	0	5	100	0	5	100
	3	7	70	3	7	70	3	7	70	3	7	70
Pneumococcic serum												
5	1	4	80	0	5	100	0	5	100	0	5	100
6	1	4	80	0	5	100	0	5	100	0	5	100
	2	8	80	0	10	100	0	10	100	0	10	100

were given five hours later. Pigeons that survived until the following morning received 100 mg. of sulfanilamide twice daily until death. No beneficial effect was noted from sulfanilamide given in this manner. There were no permanent survivals. There is one definite criticism of this method, namely, we have no evidence to offer that the sulfanilamide given these birds was absorbed. A recent publication by Bieter, Baker, Beaton, Shaffer, Seery and Orr[10] indicates that sulfanilamide administered to chickens in doses similar to those employed by us will afford blood concentrations well above 10 mg. per cent.

TABLE II

SUMMARY

Treatment	Total Number of Animals	10-12 Hours Mortality		24 Hours Mortality		48 Hours Mortality		1 Week Mortality	
		No.	%	No.	%	No.	%	No.	%
Control	30	19	63	26	86	27	90	28	93
Serum	30	1	3	4	13	10	33	11	36
Sulfanilamide	15	8	53	11	74	13	86	15	100
X-ray, prophylactic	20	11	55	17	85	19	95	19	95
X-ray therapy	20	11	55	17	85	17	85	18	90
X-ray and serum	15	3	20	4	26	7	46	8	53
Normal horse serum	10	7	70	7	70	7	70	7	70
Pneumococcic serum	10	8	80	10	100	10	100	10	100

Prophylactic Roentgenotherapy.—Irradiation was given five to six hours before the injection of organisms. In Experiments 1 and 2, 75 r. were given with a one millimeter aluminum filter at 30 cm. distance. The rays were generated at 130 Kv. 8 ma. The portal was large enough to cover the entire pigeon. Treatment lasted 1.5 minutes. In Experiment 3, 160 r. were given and in Experiment 4, 200 r. were given. No difference could be noted between the reactions of this group and the control group to inoculation by the *Clostridium welchii.* In the 20 pigeons thus treated there was only one survival at the end of the week, or a mortality of 95 per cent, in comparison to 93 per cent in the control group.

Therapeutic Roentgenotherapy.—The filter and the distance were the same as in the prophylactic roentgenotherapy group. In Experiment 1, 75 r. were given 50 minutes after inoculation; in Experiment 2, 100 r. were given 40 minutes after inoculation; and in Experiments 3 and 4, 160 r. were given 20 minutes and five hours, respectively, after inoculation. The eventual mortality in this group was 90 per cent, and no difference could be noted between the reactions of this group and the control group to inoculation by the *Clostridium welchii.* In the 20 pigeons thus treated there was only one survival at the end of the week, or a mortality of 95 per cent, in comparison to 93 per cent in the control group.

Roentgenotherapy and Serum.—Although roentgen ray alone had no effect on the infection in pigeons, it seemed possible that a higher percentage of survivals might be obtained by employing roentgenotherapy in conjunction with serum. Using the amount of serum previously described, plus irradiation, a consistently higher mortality occurred than when serum

alone was administered. In Experiment 4, 200 r. were given 29 hours before inoculation and serum was administered two hours after inoculation. In Experiment 5, 200 r. were given one hour after inoculation and the serum three hours after inoculation. In Experiment 6, 200 r. were given 45 minutes after inoculation and the serum one hour after inoculation. The mortality at the end of one week in this group was 53 per cent, while when serum alone was used the mortality was but 36 per cent.

Discussion.—Experimental and clinical evidence is somewhat contradictory on the value of the various therapeutic agents in gas gangrene. Caldwell[11] concluded that roentgenotherapy has no effect upon the progress of a fulminating type of gas gangrene, but does bring about some localization of the process when given one to two hours after inoculation, and when the animals (guinea-pigs) survive 48 hours or longer. He obtained three survivals in a group of 10 animals, while the four controls all died. In a later article, Caldwell and Cox[12] concluded, from an experimental study on some 400 guinea-pigs, that roentgenotherapy is only of slight value. However, Williams and Hartzell,[13] in two comparative clinical series of 12 cases each, had only one fatality in those patients treated with roentgenotherapy, and seven fatalities in those which failed to receive roentgenotherapy. One must remember that clinical cases of gas gangrene usually have a mixed infection, and it is possible that the beneficial effects of irradiation were due to the effect of the roentgen rays on organisms other than the *Clostridium welchii*. Against this theory are the observations of Angevine,[14] which showed that the effect of roentgenotherapy upon experimental streptococcic and staphylococcic skin infections was to increase the size of the lesions, to produce more necrosis and to enhance the invasiveness of the microorganisms.

The definite value of irradiation in gas gangrene has not as yet been proven, and statements such as: "The use of roentgen ray in treatment of gas gangrene approaches the action of a specific, in that it is by far the most effective measure so far employed," and "the use of serum is not absolutely essential to recovery and its use should be conservative, avoiding serum sickness, which only adds to the patient's difficulty" are hardly justifiable in the light of available evidence.

Bliss and Long[15] noted that sulfanilamide had a curative effect in peritonitis produced in mice by injection of *Clostridium welchii*. It should be pointed out that the infection of the peritoneum is quite different from that observed in traumatized muscles. Kendrick[16] was unable to demonstrate any marked therapeutic value of neoprontosil, sulfanilamide or sulfapyridine in *Clostridium welchii* infections produced experimentally in the muscles of guinea-pigs. In the treated group, there was a mortality of 89.5 per cent, while in the control group it was 94.3 per cent.

Reed and Orr[17] found, in experimental work on guinea-pigs, that the administration of sulfanilamide, sulfapyridine, sulfamethylthiazole or sulfathiazole showed increasing effectiveness, in the order named, in controlling infec-

tions produced by *Clostridium welchii, Clostridium septique, Clostridium sordellii* or *Clostridium novyi.* The best results were obtained when the drug was introduced directly into the wound. However, even in their experiments, it was necessary to introduce the drug before the full development of gas gangrene. Gordon and McLeod[18] also found experimentally that local administration of sulfanilamide drugs was more useful than the oral. However, they also found that sulfanilamide drugs were inferior to antisera in the prophylaxis of gas gangrene.

Sadusk and Manahan[19] demonstrated the bacteriostatic action of sulfanilamide on *Clostridium welchii in vitro.* Such action was inversely proportional to the number of organisms used in the inoculum. No bactericidal effect was noted since growth readily took place if organisms, the growth of which had been suppressed by sulfanilamide, were transferred to a sulfanilamide-free medium. They attribute Spray's[20] inability to demonstrate bacteriostatic effect of sulfanilamide to the heavy inoculation of *Clostridium welchii* used in his experiments.

Clinically, Bohlman[21] reported three cures in patients treated with sulfanilamide for gas gangrene developing following compound fracture of the lower extremity. All of Bohlman's patients, however, received some serum. Sadusk and Manahan[19] also reported two cures in postabortal *Clostridium welchii* infections with positive blood cultures. This is of some significance in view of a mortality of 85 per cent in this type of infection in patients receiving no specific therapy and 47 per cent for patients treated with specific antiserum.

Caldwell,[11] using the guinea-pig as the experimental animal, produced the counterpart of a compound fracture and infected it with a lethal number of *Clostridium welchii.* Sulfanilamide crystals implanted in such wounds after débridement seldom controlled or prevented the development of gas gangrene. However, the administration of large doses of sulfanilamide by intraperitoneal injection after the wound had been débrided, irrigated and left open, prevented the development of gas gangrene in a high percentage of instances.

Stephenson and Ross[22] compared the effectiveness of antiserum and chemotherapy and concluded from their studies that *"Cl. welchii*—Type-A—Sulfanilamide and sulfapyridine protected mice against a small number of lethal doses injected intraperitoneally as suspension of vegetative organisms in sterile soil suspension. Treatment with antitoxic serum was also effective in the case of a strain of high toxigenicity, but failed against a strain of low toxigenicity though probably of higher invasiveness. When the infected soil suspensions were injected intramuscularly, considerably more organisms were necessary to produce a fatal result and the drugs were of value only against a sublethal infection. Serum treatment was better and saved mice even from lethal doses.

"Cl. septique.—Sulfanilamide had little influence on infections of mice injected intraperitoneally or intramuscularly with suspensions of sporing

and nonsporing organisms in calcium chloride solution or sterile soil suspension. Sulfapyridine was better; in large doses it saved 50 per cent of the animals. Treatment with a single dose of antitoxic serum was at least as effective as sulfapyridine, but the best results were obtained when sulfapyridine was combined with serum, large doses of the drug being given immediately after infection and serum up to 24 hours later.

"With two strains of *Cl. oedematiens* tested so far it has been shown that neither sulfanilamide nor sulfapyridine has any influence on the course of the infection."

In considering the value of chemotherapy in clinical cases, attention again is directed to the fact that gas gangrene is very rarely a monobacterial infection. It is extremely likely that the effect of chemotherapy in improving the results in clinical cases may be due to its action on the other organisms present. In this connection, it is worth while citing the quotation from the Surgeon-General's report, as quoted by Bohlman[21]: "In Base Hospital No. 15 A.E.F., in 73 cases of gas gangrene with death, activity of the gas bacilli was self-limiting and practically confined to the first week after the wound was received. There was a drop in anaerobes from 38 to seven per cent during the first seven days, as the common pyogenes streptococcus and staphylococcus accumulated rapidly in the wound. Infections with anaerobes alone showed a high death rate but a short period of danger to life. A streptococcic bacteremia was by far the most important cause of death, especially in patients living beyond the first week, which was roughly established as a self-limiting period of gangrenous process and many deaths attributed to the anaerobes were in reality deaths due to streptococci in the process of replacing them." If a number of these deaths were due to the streptococcus, then the value of chemotherapy clinically could not be questioned.

SUMMARY

It is shown that irradiation has no demonstrable effect on *Clostridium welchii* infection in the pigeon with the dosage of irradiation used. It is shown also that irradiation used in conjunction with serum is a detriment rather than a benefit. Sulfanilamide as used in these experiments was also without value. Serum alone was of value in saving the lives of the majority of the pigeons. It is suggested that the sulfonamides may be of value in controlling the secondary infection so commonly observed in clinical gas gangrene infection.

The authors wish to express their thanks to Sharp and Dohme, who were kind enough to supply the serum used in this study.

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NONINFECTIVE GANGRENE FOLLOWING FRACTURES OF THE LOWER LEG

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GANGRENE, aside from that caused by the gas bacillus and other virulent organisms, is rare following fractures of the extremities, if one is to judge by the meager references to it to be found in the literature. Dodd,[1] in 1934, writing on this phase of traumatic surgery, collected a total of 31 cases appearing in the world literature between 1850 and 1934, among which were six cases of fracture of the lower leg followed by gangrene. Since 1934, eight more instances of this complication have been recorded. It is the purpose of this report to describe an additional case, and to consider briefly certain germane problems.

Case Report.—New York Hospital No. 286442: Male, age 23, was admitted to the New York Hospital within an hour after having been struck by an automobile. He sustained a severe crushing injury of the left lower leg which resulted in a transverse comminuted fracture of the tibia and fibula and chip fractures of the external malleolus and of the talus. There also was an avulsed wound of the soft tissues on the lateral aspect of the foot. On admission, the toes were warm and sensation intact, but the enormous hematoma about the foot and ankle precluded palpation of the dorsalis pedis and posterior tibial arteries. The popliteal artery was readily palpated, and its pulsation of good volume.

Operation: Under the effect of 120 mg. of novocain crystals injected intraspinally the wound of the lateral aspect of the foot was débrided and covered with a thin split-thickness graft. Steel pins were thrust through the tibia above and below the fractures, and anatomic reduction secured by means of a traction-distraction apparatus. The entire extremity was then encased in unpadded plaster splints lightly reinforced with circular plaster bandages.

Subsequent Course.—The patient was returned to the ward and the lower extremities elevated at an angle of 45.° Slowly, during the course of the next three days his temperature rose to 39° C., pulse 120, and his toes became deeply cyanotic. Gradually, cutaneous sensation in the toes was lost. The encasement was removed and the leg placed in a Braun-Boehler splint, with traction on the distal pin. The proximal pin, which lay well anterior to the anterior tibial artery, was withdrawn. During the course of the next two days the toes, the foot and the ankle became dusky and cool, and loss of cutaneous sensation progressed to the level of the lower calf. The calf itself was swollen, tense, and the skin shiny. This state persisted practically unchanged for the next 25 days, though the patient's vital signs returned to normal following the removal of the encasement. On the twenty-seventh day after admission a diagnosis of a large abscess of the muscles of the calf was made. On the basis of this diagnosis a long incision through the skin of the lateral aspect of the lower leg disclosed gangrenous but not infected bundles of muscles. These were smoky-white in appearance, edematous and gelatinous. All of the muscles supplied by the anterior tibial artery, namely the peronaeous longus and brevis, extensor digitorum longus, extensor hallucis longus, and tibialis anterior, were included in this process. Slowly,

these gangrenous muscles separated from surrounding viable structures; the wound became lined with granulation-tissue, and was epithelized by grafts. The tibia united firmly, and the patient was discharged six months after admission, able to walk with the aid of a brace. On discharge the anesthesia of the foot was still present.

The patient has been followed closely since discharge from the hospital, and, one year later, he is actively engaged as an iron worker, though still dependent upon his brace.

The interest in this patient lies not in the fracture but in the complication of noninfective gangrene of an isolated group of muscles. On subjecting this case to careful analysis, two features seem to be of paramount significance: (1) That the nerve supply of the lower leg and foot, which was intact on admis-

FIG. 1.—Anteroposterior and lateral roentgenograms of the tibia and fibula taken on admission.

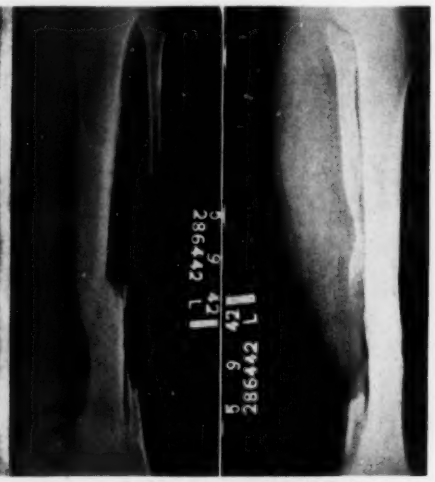

FIG. 2.—Anteroposterior and lateral roentgenograms of the tibia and fibula taken 12 months after injury.

sion, failed slowly over the course of the next two or three days; and (2) that the blood supply of the foot, which also was normal on admission, early showed signs of embarrassment followed, however, by ultimate survival of the foot. With these facts in mind, it is possible to reconstruct the course of events that led to this untoward complication. As a result of the severe crushing nature of the injury the anterior tibial artery probably was torn sufficiently to damage its intima and a thrombus slowly began to form at the site of the injury. During the course of the next few days the thrombus became so large that it occluded the lumen of this vessel. Directly dependent upon the progressive nature of this lesion are the phenomena noted clinically, namely, the intact nerve and blood supply on admission, their slow failure over the course of a few days as the integrity of the blood supply became compromised, and finally gangrene and complete loss of sensation. The ultimate viability of the foot in all probability may be ascribed to the rich collateral circulation about the ankle joint, and in the foot between the distal branches of the anterior and

posterior tibial arteries, namely, the peroneal, the dorsalis pedis and the fibular and tibial plantar arteries. Obviously, however, though this was abundant, it was inadequate to supply in retrograde fashion the domain of the proximal portion of the anterior tibial.

In an effort to establish further the validity of these assumptions, all of the reported cases of this complication, appearing in the literature from 1850 to the present time, have been critically reviewed (Table I). Analysis of these cases reveals the following interesting facts:

Age Incidence.—The average age for this group of 15 cases is 26 years. Because such a large majority of these patients fall into a relatively young age-group, degenerative diseases of the arterial wall can largely be discounted as a factor predisposing their vessels to injury and subsequent occlusion.

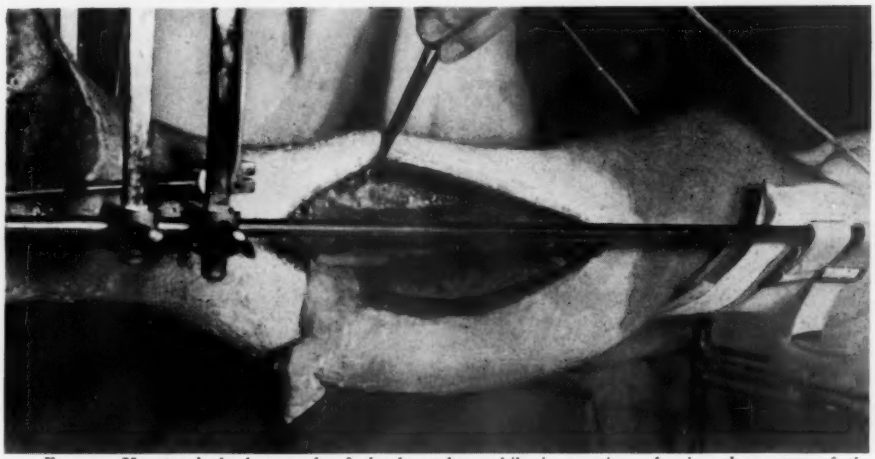

FIG. 3.—Unretouched photograph of the leg taken while in traction, showing the extent of the incision necessary to evacuate the gangrenous tissues.

Degree of Injury.—If the group be divided on the basis of severity of their injury, there are six which may be considered severe, seven moderate, and two mild. Thus, though it is apparent that gangrene as a complication is more frequently associated with the severer types of injury, the two which are classified as mild justify the conclusion that this is not invariably the case. Conversely, it is a matter of common surgical experience to see severe injuries of the lower leg unassociated with this complication.

Site and Extent of Fracture.—In the majority of the cases the fractures were extensive and involved both bones of the lower leg. There are, however, two notable exceptions, namely, the one in which the skeletal injury was merely a separation of the superior tibial epiphysis and the one in which only the fibula was broken. The sites of fracture were about evenly distributed between the upper, middle and lower thirds; some were compound, some were oblique, some transverse, and many were comminuted. It seems likely, therefore, that no particular site or type of fracture predisposes especially to this complication.

TABLE I

NONINFECTIVE GANGRENE OF THE LOWER LEG AND FOOT FOLLOWING FRACTURES OF THE TIBIA AND FIBULA—14 CASES

Case No.	Author Ref. Date	Age	Degree of Trauma	Fracture	Gangrene Extent	Gangrene Onset	Artery Occluded	Type of Fixation	Treatment and Result
1.	Schultz[1], 1897	32	Moderate	Tibia, midthird	Lower leg and foot	1st day	Popliteal		Amputation femur
2.	Jungst[1], 1879	20	Severe	Tibia, fibula at upper end	Lower leg and foot	3rd day	Popliteal		Amputation lower third
3.	Muir[1], 1924	36	Mild	Fibula, upper third	Foot	4th day	Anterior tibial Posterior tibial	Traction	Amputation lower leg and foot
4.	Gregora[1], 1927	27	Severe	Supramalleolar, tibia and fibula. Popl. hematoma	Foot	4th day	Popliteal	No incasement	Amputation lower leg
5.	Susani[2], (a), 1933	41	Moderate	Tibia and fibula, upper third	Lower leg and foot	Prior to 16th day	Anterior tibial Posterior tibial	Traction	Amputation upper tibia
6.	Susani[2], (b).	37	Moderate	Tibia and fibula, upper half	Lower leg and foot	6th day	Anterior tibial Posterior tibial	Traction	Amputation through fracture
7.	Susani[2], (c).	23	Moderate	Tibia and fibula, lower third	Foot	7th day	Posterior tibial	Traction	Amputation refused. Useless foot
8.	Susani[2], (d).	11	Severe	Tibia and fibula, upper third	Lower leg and foot	1st day	Anterior tibial Posterior tibial	No incasement	Amputation lower thigh
9.	Jones[1], 1934 (Dodd)	32	Severe	Tibia and fibula, lower half	Lower leg and foot	4th day	Anterior tibial Posterior tibial	Incasement	Amputation lower thigh
10.	Dodd[1], 1934	29	Severe	Tibia and fibula	Lower leg and foot	5th day	Anterior tibial Posterior tibial	Incasement	Amputation upper tibia
11.	Curry and Bishop[3], 1937	16	Mild	Separation of superior tibial epiphysis	Lower leg and foot	3rd day	Popliteal	Incasement	Amputation mid-thigh
12.	Fitte[4], (a), 1938	10	Moderate	Supramalleolar, tibia and fibula	Foot	4th day	Anterior tibial Posterior tibial	Traction	Amputation lower leg
13.	Fitte[5], (b).	28	Moderate	Tibia and fibula, lower third	Foot	3rd day	Anterior tibial	Cast	Amputation lower leg
14.	Ottolenghi and Spinelli[6], 1939	25	Moderate	Tibia and fibula	Lower leg and foot	4th day	Anterior tibial Posterior tibial	Traction	Amputation upper lower leg
Author's case, 1942		23	Severe	Tibia and fibula	Anterior and lateral crural M., Peroneal M.	5th day?	Anterior tibial	Cast	Function of foot fair

Extent of the Gangrene.—In nine cases this involved the lower leg and foot, in five, the foot alone, and in the present case only the lateral muscles of the calf.

Artery Occluded.—In view of the fact that all but two cases terminated in amputation, an unusual opportunity was afforded to determine accurately from the specimens where the occlusive process was located. In four, this was the popliteal; in eight, both the anterior and posterior tibial arteries were involved; in one, the posterior tibial alone, and in one, the anterior tibial alone. In this last case amputation was not performed but the gangrenous process

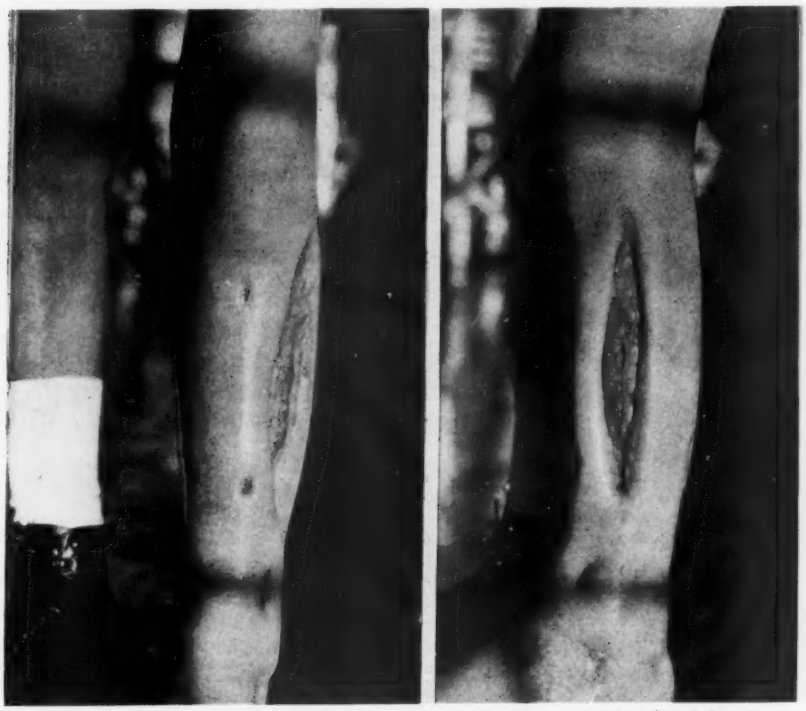

FIG. 4.—A and B. Unretouched photographs of the leg 11 months after injury.

corresponded so accurately to the areas receiving their blood supply from the anterior tibial artery that it has been assumed that it was the circulation of this artery which was compromised. There is, of course, a very definite relationship between the artery damaged and the extent of the gangrene. The reasons for vascular damage in these injuries are discussed below.

Method of Treatment of the Fracture.—This problem was reviewed with great interest, for during the hospitalization of the patient the opinion was voiced that the encasement might have been the cause of the complication. Although it is impossible to refute this indictment of the encasement, it was found out that of the 13 cases in which data were available, six had been treated with traction, five with encasements, and two, without either of these

aids to fixation. It seems justified, therefore, to postulate that the complication, in all probability, was not related to the method employed for immobilization.

Results.—In all but two cases amputation became imperative; five through the thigh and eight through the lower leg. The two exceptions are one case which refused amputation, and was left with a useless leg, and the present case left with a foot functioning only partially even when supplemented by a brace. There were no deaths in this series of cases.

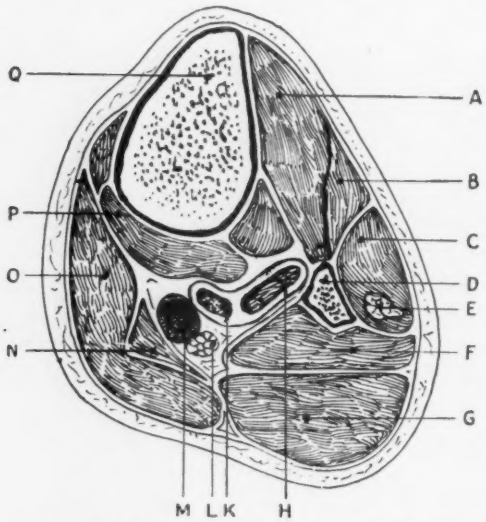

FIG. 5.—Photographic section through the upper third of the right leg at the level of the bifurcation of the popliteal artery; this is at the origin of the anterior tibial artery, as it arises from the termination of the popliteal artery and runs horizontally forward to the anterior compartment of the leg in close proximity to the fibula.
(Dodd, Harold: Brit. Jour. Surg., **22**, 246, 1934-35). (*Courtesy British Journal of Surgery*)

Key—
A. Tibialis anticus.
B. Extensor digitorum longus.
C. Peroneus longus.
D. Fibula.
E. External popliteal nerve.
F. Soleus (fibular head).
G. Gastrocnemius.
H. Anterior tibial artery.
K. Termination of the popliteal artery and the beginning of the posterior tibial artery.
L. Internal popliteal nerve.
M. Internal popliteal vein.
N. Soleus (tibial head).
O. Gastrocnemius.
P. Flexor longus digitorum, with tibialis posticus anterolaterally.
Q. Tibia.

DISCUSSION.—In considering the cases of gangrene following fractures of the lower leg from the point of view of the etiology of the complication, the most important single factor seems to be the damage sustained by the blood vessels of this portion of the lower extremity. Dodd, in his survey of the subject, calls attention to the fact that the bifurcation of the popliteal artery is particularly vulnerable because of its anatomic relationships. He states: "This is anchored by the fibrous arch of the soleus, by the passage of the anterior tibial artery over the interosseous membrane, by its proximity to the fibula and by the origins of small anastomotic branches to the knee joint.

For the cross-section through the termination of the popliteal artery I am indebted to Professor Harris, of University College Hospital. He very kindly sectioned a leg, which was photographed; such a diagram is, I think, unobtainable from the textbooks. He pointed out the comparatively intimate association of the bifurcation with the neck of the fibula. He showed me radiograms demonstrating on the inside of the neck of the fibula a slight groove which is made by the anterior tibial artery as it passes from behind to the front compartment of the leg. This emphasizes the vulnerability of the artery to injury in fractures of this region of the fibula."

In other words, the origins of the anterior and posterior tibial arteries are so rigidly fixed by the surrounding structures that they are subject to injury not only by direct violence, but also by force transmitted to their bifurcation from other parts of the leg. For instance, in Case 3, a fracture of the fibula alone was sufficient so severely to damage both of these vessels that gangrene of the foot occurred. Further, Dodd insists, with real justification, that not only will complete division, penetration or partial rupture of the artery be sufficient to effect occlusion, but also that contusion or rupture of the intima alone will result in occlusion of the vessel with thrombus forming at the site of such intimal damage. He also points out that segmental spasm due to injury may lead to gangrene if allowed to persist for any great length of time. The present case, as well as others recorded, tend to support the claim that any fracture of the lower leg may be associated with damage to the arteries of sufficient severity to impair the circulation to such a degree that the complication of noninfective gangrene occurs.

Treatment.—Gangrene, once it has occurred, is amenable to only one therapeutic procedure, namely, amputation. The logical approach to this complication, therefore, is the early recognition of threatened gangrene and the prompt adoption of methods of treatment which might reasonably be expected to prevent its development. If in every case of fracture of the bones of the lower leg the possibility of gangrene were borne in mind, this complication might perhaps be averted. A diagnosis of impending gangrene will not likely be made if it is not considered as a possibility. Particularly should fractures of the lower leg be watched for evidence of failing arterial pulsation, progressive loss of sensation, coldness, increasing cyanosis and pain. Should any one or any combination of the signs or symptoms appear, a progressive thrombotic process must be suspected. Adequate heparinization of the patient should be strongly considered as a method of preventing further thrombosis. A wide incision should be made in the neighborhood of a large hematoma to relieve pressure and to explore for an injured vessel. If possible, such a vessel should be repaired by end-to-end suture and even perhaps by a free venous graft. Some form of sympathetic block must be instituted. A plea is made for the principle of "look and see" rather than "wait and see." Since most of the cases presented an interval of time of from one to sixteen days before gangrene occurred, some warning of the imminence of the complication is given. In the present case the gangrenous process probably did not start until about the third day. If the serious nature of the complication had been anticipated, it is possible that it might have been avoided by the institution of one or more of the measures outlined above.

SUMMARY

All reported cases of noninfective gangrene following fractures of the lower leg have been reviewed and tabulated.

An additional case has been recorded in detail.

The anatomic relationships of the division of the popliteal artery into the

anterior and posterior tibial arteries have been found to be the predisposing factors in this complication.

Methods of diagnosis and treatment of impending gangrene have been suggested, in order to prevent this process from reaching an irreversible end-point.

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RECURRENT ULCERATION FOLLOWING SUBTOTAL GASTRECTOMY IN THE TREATMENT OF GASTRODUODENAL ULCER*

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ALTHOUGH subtotal gastrectomy has been employed in the treatment of gastroduodenal ulcer for more than two decades, it has not been possible, as yet, to discuss its asserted effects upon an adequate factual basis. The usefulness of the operation has been ascribed to its efficacy in preventing the faults of more conservative measures, particularly those of gastro-enterostomy. Specifically, the proponents of subtotal gastrectomy have claimed that its radical approach not only minimizes the incidence, but also averts the serious complications of subsequent ulceration. Up until now, attention has been principally focused upon the first of these contentions. Despite ceaseless debate and discussion, no agreement exists concerning the frequency of renewed ulceration following other operations. Moreover, as patients have been observed to develop recurrent symptoms after remaining well for periods as long as 30 years, it has become increasingly evident that any contention as regards a therapy's efficacy to prevent reactivations of the disease can carry little weight, unless it is supported by the results observed in the following of cases over extended periods of time. Unfortunately, no method has as yet been devised for determining how long patients must be observed to permit conclusive inferences on that score. Time and experience have proved that the commonly employed standard of a five-year follow-up is insufficient, at least for evaluating the results of any therapy for duodenal ulcer, and there is much doubt concerning the validity of conclusions based upon ten-year studies. Up to date, there have been few reports, if any, with the employment of subtotal gastrectomy which fulfill these questionable criteria. Its complicating incidence of recurrent ulceration, therefore, awaits the results of further experience and the development of a satisfactory method for appraising them.

The second claim made for subtotal gastrectomy relates to its efficacy in averting the serious complications of peptic ulcer. Although this contention lends itself to more definitive discussion, it has received relatively little critical consideration. This has been due, in part, to the limited opportunity to study the operation's late effects. In view of these facts, it was thought that it might be of value to report and discuss the clinical features of the cases that are known to have developed symptoms and evidence of recurrent ulceration

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after the employment of subtotal gastrectomy for duodenal and gastric ulcer in the Gastric Clinic of Mount Sinai Hospital, New York, N. Y. These cases, which have been detected in following a significant number of patients for varying lengths of time during the past 17 years, contribute a factual perspective to an aspect of the operation, which, thus far, has been so speculative.

In recent years the status of some of the problems requiring consideration in discussing the data of this report has changed so materially that it is necessary, for purposes of clarity, to indicate the particular point of view that is held concerning them. In the first place, there has been much confusion as to what is meant by subtotal gastrectomy. While different interpretations have been given to the rôle which certain factors play in contributing to its success, *e.g.,* extent of resection, method of restoring continuity, removal of lesion, *etc.,* it is well agreed that the basic prerequisites of the operation are the removal of the pylorus, a radical resection of all antral tissue, and the excision of the ulcer. Since operations involving limited resections, *e.g.* pylorectomy or the prepyloric exclusion, which retains the pylorus itself, do not fulfill these prerequisites, cases of those categories were not included in this report. Likewise, operations that employ entero-anastomoses were omitted, since that procedure tends to divert the alkaline intestinal contents and thereby to defeat a major objective of subtotal gastrectomy—the influencing of the gastric acidity. The required extent of resection is not easy to define. This clinic has employed a technic, originally devised by A. A. Berg,[1] in which the proximal level of resection reaches well above the reentrant angle of the stomach; gastro-intestinal continuity being restored by a partial terminal-lateral anastomosis (Hofmeister method). Until three years ago the retrocolic anastomosis was preferred, so that the results in this report relate particularly to that method.

The employment of subtotal gastrectomy has been so closely linked with the problem of gastrojejunal ulceration, that the principal interest in any report of its results centers about that question. A consideration of its significance is particularly pertinent to the purposes of this discussion. Gastrojejunal ulceration is generally regarded to be an induced condition, which results from the anastomosing of the jejunum to the stomach. While primary ulcers of the jejunum have been reported, they are so rare that, for all intent and purposes, they may be considered nonexistent. Various theories have been suggested for the causation of anastomotic ulcer, but none have proven adequate, since it has been found to occur after every suggested technic and despite every precaution. While it may follow any operation which joins the stomach to the jejunum, it is observed almost exclusively in ulcer disease, and only in the presence of free hydrochloric acid. This communication agrees with the current opinion which considers gastrojejunal ulcer to be caused by the factors essentially responsible for ulcer disease itself, and that it is, therefore, a recurrent peptic ulceration. As such, its effects are no different from those seen with an ulcer of the stomach or duodenum, *e.g.,* pain, hemorrhage, perforation, obstruction, *etc.* It is because of anatomic

factors that the complications resulting from the activity of anastomotic ulceration tend to be more serious and more difficult to treat.

Inasmuch as this report represents the experience of a particular clinic, certain factors relevant to it must be appreciated. In the first place, it relates to patients who had been operated upon between 1923 and January 1, 1940 on the Gastric Ward Service of Mount Sinai Hospital, New York, N. Y. The results reflect the experience of an entire staff of trained gastric surgeons rather than any one individual surgeon. The patients were preponderantly Jewish, and were chiefly factory workers and tradesmen. There were relatively few artisans and clerical workers, and practically no agricultural workers. The indications for operation were, as a rule, failure to respond to adequate medical treatment, *e.g.,* intractable pain, unrelieved obstruction, and repeated hemorrhage. The cases, comprising this study were observed in the regular Follow-up Clinic, which is conducted bimonthly before a group of surgeons, gastro-enterologists, and house staff. All observations are made through personal interviews, and are the result of a combined rather than an individual opinion.

The symptomatology, pathology and diagnostic features of recurrent ulceration, which in our opinion includes gastrojejunal ulcer, have been fully described in numerous reports and need no special consideration. The term "ulcer" and "ulceration" are used interchangeably as descriptive of the "peptic ulcer" in the pathologic sense. It is frequently difficult and often impossible, clinically, to determine the exact cause of complaints after gastric operations. Although roentgenologic investigation reveals accurate confirmatory evidence of recurrent ulceration in a high percentage of examinations, there are instances in which the findings are negative. These cases cannot be differentiated with certainty from those with "gastritis" or "erosions," which give rise to epigastric pain and bleeding. This study, therefore, has been limited to those patients with complaints after subtotal gastrectomy, who had manifested unequivocal or convincing presumptive evidence of recurrent ulceration. This evidence will be discussed presently.

While, surgically, a recurrence of the disease is the most important cause of failure after radical resection, it must be remembered there are other factors which may contribute to an unsatisfactory result. For example, about 10 per cent of the patients manifested a train of symptoms, which seemed to be caused by some disturbance in physiology, resulting from the operation itself rather than any activity of ulcer disease. Some had symptoms of a vagotonic type, *e.g.,* sweating, nausea, palpitation, weakness; others complained of failure to gain weight, and a small number suffered from periodic diarrhea. While these symptoms were disturbing, for the most part, they were not incapacitating. There was another group of ten patients with so-called dyspeptic complaints, *e.g.,* heart burn, sour eructations, who contended they were not improved by the operation. Repeated examinations have failed to reveal any organic cause for their complaints, and they, unquestionably, represent a neurotic group, such as is encountered in every field of surgery.

TABLE I

SUBTOTAL GASTRECTOMIES (1923–1940)

Type of Case	Operative Survivors	Recurrences	Basis for Diagnosis: Operation	Gastroscope X-ray	Gross Bleeding
I. Duodenal Ulcer					
(a) Primary*	366	27	6	16	5
(b) Secondary†	136	13	7	4	2
(c) Total	502	40	13	20	7
II. Gastric Ulcer	98	1	0	1	0

* Primary case—no previous gastric operation.
† Secondary case—one or more previous gastric operations.

A summary of the data, upon which this study is based, is given in Table I. It will be noted that 41 cases are classified as recurrent ulcerations. The diagnosis was definitively established by operation* or by autopsy in 13 cases. In 21 of the remaining 28 the diagnosis was confirmed roentgenologically or gastroscopically. There were seven patients who returned with severe, painless, gross hemorrhage (melena or hematemesis) at intervals varying from a few months to several years after operation. Although the roentgenologic examinations in these cases were negative, it is not unlikely that recurrent ulcerations were responsible for their bleeding. There are well-founded reasons for such an assumption. In the first place, we know from our experiences with the employment of subtotal gastrectomy in cases of "painless bleeding" following gastro-enterostomy, that the resected specimens frequently reveal healed or superficially healing gastrojejunal ulcerations.[2] In the majority of these instances, the roentgenologic examinations are negative, but it must be borne in mind that investigative examinations and surgery are usually carried out after cessation of the bleeding and after prolonged medical treatment. It is not usual to observe similar roentgenologic and pathologic findings in cases of primary duodenal ulcer associated with otherwise symptomless bleeding after comparable preoperative treatment. Moreover, there have been several patients in this clinic in whom operation had been limited merely to exploration because of the paucity of findings. At subsequent celiotomy, because of recurrent symptoms, large active duodenal ulcers were found. There is no doubt, therefore, that a peptic ulcer may give rise to painless hemorrhage, and that negative roentgenologic findings do not controvert that diagnosis. This presumptive evidence of recurrent ulcer must be weighed against the possibility of erosions and postoperative gastritis as causes for painless bleeding. This aspect of that problem has been fully considered by Moschcowitz, Mage and Kugel.[3] There is a well-recognized school which regards erosions and gastritis to be manifestations of ulcer disease, particularly when

* Operative findings indicating active or healed ulcers were regarded to be adequate evidence of such lesions. In two instances penetrating jejunal ulcers were encountered, but not removed, so that specimens were not available for examination. In nine cases specimens were available. Seven disclosed ulcers. No evidenced ulceration was found in two, in which the surgical findings were considered to be those of healed lesions.

they occur in individuals who are known to have had peptic ulcer. In a sense, it seems to be an academic question, whether a severe hemorrhage, occurring months or years after subtotal gastrectomy, arises from an ulcer or an erosion. In the final analysis, the operation did not protect the patient against a serious complication of ulcer activity.

There were four patients who returned with recurrent symptoms at intervals of six months, two, six and seven years after their operations. Their roentgenologic examinations disclosed small, tender pockets, which appeared to be in the stomach proper, near the stoma, and in the region of the lesser curvature. One of these cases was the only recurrence detected after subtotal gastrectomy for gastric ulcer, in this clinic during the past 17 years. The patient was a male, age 58, who was subjected to operation after one year of severe postprandial pain. The resected specimen revealed a small lesser curvature ulcer and a severe chronic gastritis. Within six months there was a return of epigastric pain and vomiting. Roentgenograms revealed a small, irregular penetration, suggesting a lesion in the postoperative stomach. This patient was lost to observation after 18 months, but during that time he had several recrudescences of pain and vomiting. The other three cases represent the development of gastric ulcers after the employment of subtotal gastrectomy for the treatment of duodenal ulcer. A newly formed ulcer of the lesser curvature of the stomach is not an infrequent cause for failure after the performance of a gastro-enterostomy for an ulcer of the duodenum. In the remaining 37 cases in this group, the diagnosis of a gastrojejunal ulcer was established, definitively, by operation, roentgenologic and gastroscopic examination in 30 patients, and, presumptively, by the occurrence of "gross hemorrhage" in seven. In view of the solitary recurrence after gastric ulcer, this study resolves itself into a discussion of 40 failures following the employment of subtotal gastrectomy for primary and secondary duodenal ulceration.

There were 44 female patients who survived a subtotal gastrectomy for primary or secondary duodenal ulcer; of these, two are known to have developed gastrojejunal ulcers. One case was of particular interest, in that she developed her first symptoms at the age of 60. After eight months of ineffectual medical therapy, she was subjected to a radical resection, for a penetrating ulcer of the duodenum. Within three months there were severe recurrent symptoms which remained intractable to conservative measures, and necessitated reoperation within a year. At exploration, a large perforating jejunal ulcer was found. The second case was a young woman, age 19, who had experienced ulcer symptoms, including hemorrhage, for three years. Four years following her subtotal gastrectomy she returned with an alarming gross hemorrhage, which had reduced her hemoglobin to 25 per cent. It is asserted that gastrojejunal ulcer rarely occurs in the female; and when it does, it appears late in life. It is interesting that one of these patients was among the most severe, and the other was among the youngest in this whole group of failures.

TABLE II

TIME OF ONSET OF POSTOPERATIVE RECURRENCE

	Primary and Secondary Duodenal Ulcer			Gastric Ulcer		
Years Postop.	Surviving Cases	Followed Cases	Recurrences	Surviving Cases	Followed Cases	Recurrences
1 yr.	502	453	15	98	95	1
2 yrs.	465	385	9	95	82	—
3 yrs.	421	330	6	87	69	—
4 yrs.	411	280	4	79	56	—
5 yrs.	397	250	0	76	52	—
6 yrs.	382	218	1	72	45	—
7 yrs.	364	211	2	64	39	—
8 yrs.	339	179	0	58	34	—
9 yrs.	315	152	0	54	34	—
10 yrs.	274	128	2	54	31	—
11 yrs.	249	113	0	49	26	—
12 yrs.	220	97	1	40	17	—
13 yrs.	174	75	0	29	11	—
14 yrs.	137	53	0	20	7	—
15 yrs.	86	33	0	15	6	—
16 yrs.	58	21	0	4	2	—
17 yrs.	17	8	0			

Time of Onset of Recurrent Symptoms.—Table II indicates the number of surviving cases that potentially might have been followed for each of the 17 years of observation; the number of patients that actually was followed; and the postoperative year in which the various recurrences were detected. It will be noted that the renewed ulcerations occurred at intervals varying from less than a year to 12 years after operation. The fact that six of the 40 failures developed after the fifth postoperative year, serves to emphasize the inadequacy of a "five-year" follow-up for appraising results following the employment of subtotal gastrectomy for duodenal ulcer. These late recurrences, moreover, were observed during the period when an increasing percentage of patients became lost to follow-up observation. The meaninglessness of publishing "three- or five-year 'cures' for gastro-enterostomy" was strikingly demonstrated by St. John, *et al.*,[4] in their recent study of results of treatment for peptic ulcer. The above observations are of particular significance since they not only illustrate the uncertainty of cure, but they also emphasize the absolute need for following patients indefinitely, in order to evaluate properly the efficacy of any ulcer therapy.

Symptoms.—The symptomatology in this group of recurrences was not essentially different from that observed with primary peptic ulcer, except for their greater severity and intractability. In 23 cases, pain was the only symptom; in six instances the pain was associated with bleeding, and in 12 patients there was gross hemorrhage (melena or hemetemesis) with little or no pain. The clinical courses of these patients have been variable. The symptoms became severe enough to necessitate reoperation in 12 patients; ten in this institution and two elsewhere. All these operated cases were found to have gastrojejunal ulcerations. In two instances, the cicatrizing effects of the lesion had caused low-grade partial obstruction; in two other cases, the edema and swelling resulting from active penetration into the adjacent mesen-

tery of the small intestine had produced more acute obstructive symptoms. In one instance, there was a free perforation of a jejunal ulcer, which necessitated an immediate operation. Another patient (unoperated) died from the effects of a gastrojejunocolic fistula, which was confirmed by autopsy. There are three patients under observation at present, who have been readmitted to the hospital three, five, and nine times, respectively, during the past six years, because of the severity of their complaints. Except for the patients with painless bleeding, who have responded satisfactorily to conservative treatment, the remaining cases have been controlled only with fair success by similar measures.

Hemorrhage.—Gross bleeding (melena or hematemesis) was a prominent and serious occurrence in 18 of this group of 41 recurrent ulcerations. The full details of these cases and their implications have been discussed in a previous communication by Colp and the author.[5] These hemorrhages occurred at intervals of a few months to 12 years after operation, and varied from a moderate to a severe degree. Ten of these patients bled prior to their subtotal gastrectomies; six had repeated episodes. Five of these six patients with multiple hemorrhages also bled several times after operation. There were six cases in which the bleeding was associated with pain; all were found to have jejunal ulcerations; two by operation, and four by roentgenographic evidence. There were 12 cases in which bleeding was the only symptom. All these patients responded to medical therapy. Roentgenologic examinations, made after bleeding had stopped, were negative in seven instances; in three, jejunal ulcers were reported, and in two, lesser curvature defects were noted.

These observations are somewhat at variance with the general impression which exists concerning the particular efficacy of subtotal gastrectomy in the problem of bleeding. The fact that a majority of patients who bled after operation also bled prior to it, may suggest the possibility of a bleeding diathesis. However, it seems far more likely, from the character and history of the clinical material, that the hemorrhages were merely the results of recurring ulcerations or "erosions." While subtotal gastrectomy may not provide a certain assurance against subsequent bleeding, it finds its particular field of usefulness in the problem of the actively bleeding ulcer, because its radical approach entails a direct attack on the lesion. As a consequence, it is far more likely to succeed than is the indirect approach of gastro-enterostomy. However, once the bleeding of a duodenal ulcer is arrested, whether it be by the conservative or the radical operation, a subsequent hemorrhage at a late interval is, as a rule, caused by a gastrojejunal ulceration and not by the original lesion. It is well to remember that this holds true for a gastroenterostomy performed for duodenal ulcer in its nonbleeding stage.

In reviewing this series of 41 recurrences after subtotal gastrectomy, certain facts stand out clearly. In the first place, there is the striking difference in response of gastric and duodenal ulcer to the same operation. The reason for this difference remains unsolved, and its answer may contribute much

to a better understanding of the ulcer problem. The second significant observation pertains to the question of the relationship between gastric acidity and recurrent ulceration. Up to date, this clinic has not observed a recurrent ulcer in the absence of free hydrochloric acid. Acidity studies[6] have revealed that subtotal gastrectomy creates an achlorhydria in at least 90 per cent of patients with gastric ulcer, and it produces an apparent achlorhydria in about 50 per cent of patients with duodenal ulcer. It is in this group with unneutralized free hydrochloric acid, that renewed ulceration is encountered. It is to be noted that the observations relating to acidity and those pertaining to recurrent ulceration after radical resection for duodenal and gastric ulcer are closely correlated. These facts not only emphasize that the effect of radical resection to a large extent is due to a modification of the acid factor in gastric digestion, but they also lend much weight to the concept which postulates that an achlorhydria is the best assurance against subsequent ulceration.

Most proponents of subtotal gastrectomy regard the creation of an innocuous achlorhydria to be one of the principal objectives of the operation. Some have contended that if such an effect is not obtained, or if recurrent ulceration develops, it is presumptive evidence of the inadequacy of the extent of resection. Such a doubt naturally arises with every unsuccessful case. However, it must be remembered that as long as any stomach tissue containing parietal cells remains, free hydrochloric acid may be secreted. Experience has shown this to be so. There are certain cases, of which there were two in this series, that continue to have free hydrochloric acid in their gastric contents, in spite of repeated resections which leaves but a minimum of stomach. Some of these patients, presumably, possess an ulcer diathesis that is so severe that ulceration recurs, despite every effort to prevent it. While radical resection is essential, it is not the deciding factor which determines either a gastric achlorhydria or the prevention of renewed ulceration. The difference in response of gastric and duodenal ulcer to the same extent of resection is evidence of that fact.

Recently, certain observers have stressed the removal of the ulcer as a *"sine qua non"* for insuring the success of subtotal gastrectomy. It is interesting to note that 30 of the 41 resected specimens contained the ulcer, so that in this particular experience the excision of the lesion did not safeguard patients against subsequent failure.

It is evident from the manifestations of this group of recurrent ulcerations, *e.g.,* uncertainty of occurrence, intractability of pain, perforation, repeated hemorrhage, and gastrojejunocolic fistula, that a patient who is subjected to subtotal gastrectomy is not rendered immune to the serious complications which are noted after other gastric operations. While the likelihood of such occurrences appears to be negligible after its employment for a lesser curvature ulcer of the stomach, it is not insignificant in the case of duodenal ulcer. This failure of subtotal gastrectomy to fulfill completely its anticipated effects in the surgical treatment of duodenal ulcer, does not, itself, negate contentions

as regards its relative merits. This clinic regards radical resection to be the best available operation for duodenal ulcer, but it is the present impression that it leaves much to be desired. At the outset of this report, we indicated the impossibility of drawing logical statistical conclusions concerning the incidence of recurrence under prevailing conditions. To do so is only to create new fallacies and to perpetuate old ones. In our opinion[7] the statistical confusion which exists concerning the effects of various treatments for peptic ulcer cannot be settled until some generally accepted logical method is devised for studying results in a uniform way, and with a particular provision for the manifold variables which make for unwarranted actual and comparative inferences. It will be noted that, in Table III, the minimal known percentage-incidence of recurrence is recorded. It was determined by computing the known number of recurrences against the total number of surviving patients, and merely means that, in the experience of this particular clinic with the employment of subtotal gastrectomy, the percentage-incidence of recurrent ulceration cannot be less, but may be more than the indicated figures. These results do not permit any valid comparative inferences. To utilize them for such purposes would be misleading.

TABLE III

MINIMAL INCIDENCE OF RECURRENCE

Type of Case	Operative Survivors	Recurrences (Operation + X-Ray)	Recurrences + Gross Bleedings
I. Duodenal Ulcer			
(a) Primary Cases	366	22 (6%)	27 (7.4%)
(b) Secondary	136	11 (8%)	13 (9.6%)
(c) Total	502	33 (6.6%)	40 (8%)
II. Gastric Ulcer	98	1 (1%)	1 (1%)

SUMMARY

In this communication certain observations have been presented which tend to shed a significant factual light upon the effects of subtotal gastrectomy in the treatment of duodenal and gastric ulcer. There has been no intention to support or refute any contentious point of view, but an attempt has been made to draw attention to certain facts, which may contribute to a better understanding of the *modus operandi* of subtotal gastrectomy, and the nature of its complications.

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PRIMARY CARCINOMA OF THE DUODENUM

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PRIMARY CARCINOMA OF THE DUODENUM is a very rare condition, having a reported incidence of from .03 to .003 per cent of all autopsies, or, in other words, from one case in 3,000 to one in 31,000 autopsies.[16, 21, 27, 31] While an extensive literature has accumulated on this subject, critical analysis of the cases reported demands that many, especially among the earlier papers, must be discarded for the lack of pathologic proof of the diagnosis. We wish to present a proven case of primary carcinoma of the third (infrapapillary) portion of the duodenum, and to briefly review the literature in order to emphasize the more important clinical facts and to bring up to date the statistics relative to the incidence and the results of surgical therapy.

Case Report.—Hosp. No. 243817: H. B., white, male, age 53, was admitted to the Jewish Hospital, August 14, 1941, with pain in the entire right side of the abdomen for 3 mos., radiating to the back, occasionally relieved by food; constipation 6 mos.; weakness and anorexia, 3 mos.; loss of 20 lbs. in 3 mos. The pertinent physical findings were pallor, evidence of recent weight-loss, tenderness in the right loin, right lower quadrant, and right costovertebral angle. All laboratory data were normal except for a R.B.C. count of 3,860,000, Hb. of 56 per cent, and occult blood in the stool. Roentgenograms of heart, lungs, and kidneys were normal; a shadow on a plain film of the abdomen was suspicious of gallstones. G.I. series revealed an irregular duodenal cap on most films, judged to be spastic. Barium enema showed some irregularity of the cecum. Gastroscopy, 8/24/41, was negative. *Preoperative Diagnosis:* Malignancy of the cecum and cholelithiasis.

Celiotomy, September 5, 1941, revealed a generalized abdominal carcinomatosis, but no intrinsic lesion was observed in any organ. Infiltrated para-aortic nodes were palpated all the way up to the root of the mesentery. The gallbladder contained calculi. An omental nodule, removed for biopsy, was reported as metastatic adenocarcinoma, origin unknown. The subsequent course was uneventful until September 18, 1941, the 12th postoperative day, when, while out of bed, the patient complained of sudden severe generalized distress, and died within five minutes. The clinical impression of the cause of death was coronary occlusion.

Necropsy.—No. 41-168. Dr. I. Roy Gold: The principal findings were generalized arteriosclerosis, with a fresh coronary thrombosis; carcinoma of the infrapapillary portion of the duodenum, with metastases to the lymph nodes, pancreas, liver, mesentery and peritoneum; cholelithiasis; urolithiasis (bladder); and a small leiomyoma of the stomach wall. The duodenal neoplasm (Figs. 1 and 2) almost completely encircled the bowel, beginning 1.5 cm. beyond the opening of the common bile duct (which in this case was separate from and 2 cm. distal to the opening of the duct of Wirsung), and extended caudad for 6 cm. The mucosa in the base of the lesion was excavated in a Y-shaped area but was intact over its raised, firm, pearly-white edges. Both ducts and the papilla were entirely uninvolved; and the head of the pancreas was secondarily invaded. Posteriorly, the lesion was continuous with a large mass of similar tumor tissue which replaced the para-aortic lymph nodes. The microscopic appearance was typical of cylindrical cell carcinoma of intestinal origin (Fig. 3).

COMMENT.—While the diagnosis of gastro-intestinal malignancy was made in this case before operation, its duodenal origin was not determined either clinically or roentgenographically, or even at exploration. Similar errors are common among reported cases, for several reasons. The symptoms are often vague or obscure, and, as in this case, fail to point to any specific level. When signs of intestinal obstruction are present, the condition is usually mistaken for ulcer or gastric neoplasm, while jaundiced cases are commonly

FIG. 1.—Drawing of the gross specimen as opened from behind. Probe is in the opening of the duct of Wirsung, which did not join the papilla of Vater in this case. The common duct and papilla have been opened, the latter appears midway between the duct of Wirsung and the upper margin of the tumor. Inset shows the lesion and ducts in their anatomic positions.

mistaken for carcinoma of the head of the pancreas or for intrinsic common duct obstruction. The location of the primary site at operation may be difficult in the distal retroperitoneal duodenum, as in the present case, where the cancer, though large, was obscured by the massive retroperitoneal lymph node involvement around it as well as by the root of the mesentery.

However, the chief reason for error is the fact that duodenal carcinoma, because of its rarity, is usually not consciously considered. In this case the lesion was plainly to be seen on the roentgenograms, when they were reviewed, to be in the distal duodenum (Fig. 4). The roentgenographic appearance of the condition has been well described by Hoffman and Pack,[21] Howes,[23] Doub and Jones,[14] and Weintraub and Tuggle.[47]

Primary duodenal carcinoma is anatomically subdivided into supra-papillary, peripapillary and infrapapillary tumors on embryologic, patho-

logic, as well as upon clinical grounds. The suprapapillary portion is above the common duct opening into the duodenum; it is derived from the foregut. The infrapapillary portion arises from the midgut (yolk sac) while the region of the papilla is in the zone between the two. In the latter area, carcinoma may arise from one of several of the epithelia in the region (duodenum, ampulla of Vater, terminal bile duct, or terminal pancreatic duct), while in the other areas, the duodenal mucosa is the sole offending epithelium.

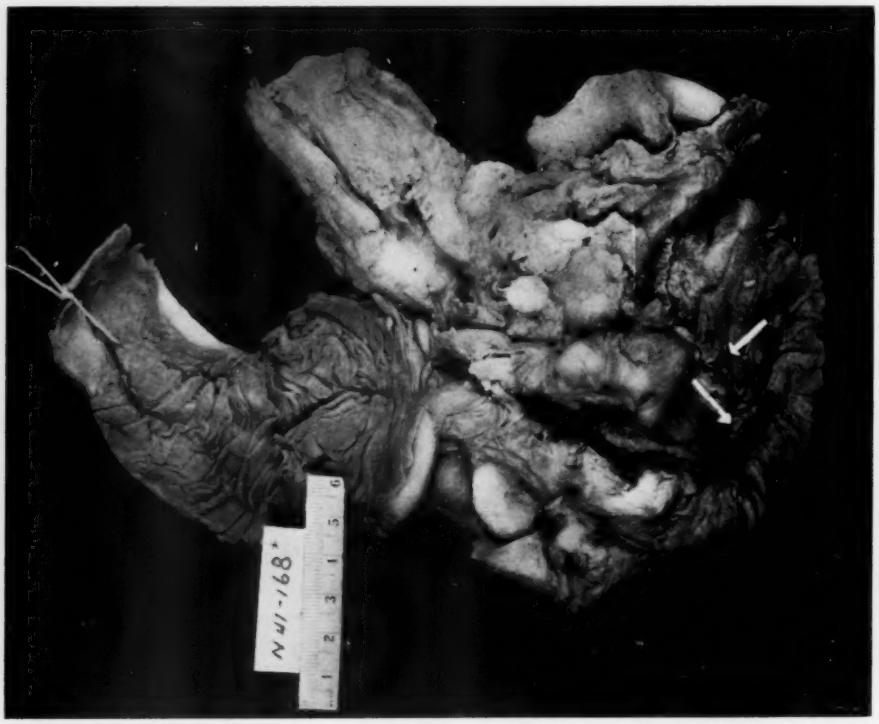

FIG. 2.—Photograph of gross specimen as opened from behind. Upper arrow points to opening of duct of Wirsung; lower arrow to papilla of common duct. The lesion is surrounded by enlarged, invaded retroperitoneal lymph nodes, and above, by the pancreas, also secondarily invaded.

Symptomatology and surgical therapy also vary with the different locations.

The literature of all three groups was very carefully analyzed in a series of three articles by Stewart and Lieber,[45] and Lieber, Stewart and Lund,[30, 31] up to 1937, for supra- and infrapapillary growths, and, up to 1939, for peripapillary tumors. These three papers reviewed a total of 565 previously reported cases, of which only 298 were acceptable as authentically proven, to which the authors added 25 of their own, making a total of 323 acceptable cases. We have found 80 additional cases in the literature to date, of which 62 were acceptable. This number, plus the present case, added to the above figure aggregates 386 known proven cases to date.

Of these, 77, or 19.9 per cent, were suprapapillary; 250, or 65 per cent, were peripapillary; and 58, or 15 per cent were infrapapillary. The distribution of the three types, as reported by other authors from smaller series, varied somewhat, but not radically from our figures.[14, 21, 48]

While the over-all incidence of primary duodenal carcinoma, as mentioned above, is from .03 to .003 per cent, Hoffman and Pack[21] estimated that it comprises 0.3 per cent of all intestinal carcinomata. They also report that in a series of 228 reported cases of small intestinal carcinoma, 45.6 per cent occurred in the duodenum. Most cases occur in the sixth decade of

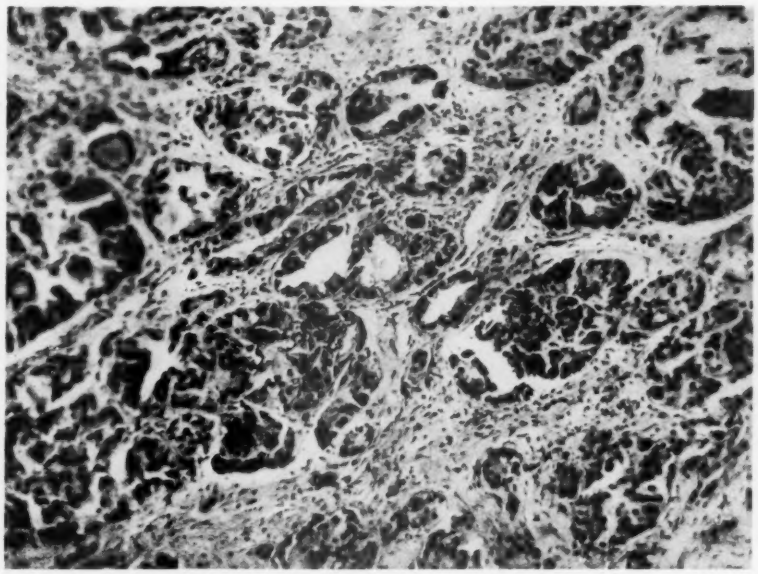

FIG. 3.—Photomicrograph of the general histologic picture. The cells are irregular in size and shape and are arranged in acinar fashion; they invade the muscles and lymphatics of the duodenal wall. (× 200)

life. In the larger series reported, males predominated over females, in ratios varying from 2:1 to 4:1.[21, 30, 31, 45]

In the review below, certain clinical highlights of the disease are stressed, especially the more recent contributions. For detailed analyses of all phases of the subject, the reader is referred to the excellent articles of Stewart and Lieber,[45] Lieber, Stewart and Lund,[30, 31] Hoffman and Pack,[21] and Cooper.[12]

CARCINOMA OF THE SUPRAPAPILLARY PORTION

Stewart and Lieber[45] analyzed 104 reported cases, accepted 57 as authentic, and added six of their own, making a total of 63 proven cases up to 1937. We have analyzed 15 additional cases, 14 of which are acceptable, bringing the present total of proven cases up to 77. The new cases are: Three reported by Hoffman and Pack;[21] two by Cace;[9] and one each by Jerrel,[25] Woods,[48] Allen,[1] Hart Hansen,[20] Nicolini,[39] Bolo, Jakob and

Busch,[3] Wiig,[51] Masciottra,[35] and Bonarino-Udaondo.[4] Another case of Hart Hansen's,[20] was discarded for lack of pathologic proof.

The chief symptomatology of all malignant tumors of the duodenum is that of duodenal obstruction, which varies more with the degree of obstruction than with the location of the growth in the duodenum (Eger[16]), except that periampullary carcinoma produces jaundice much earlier than those of

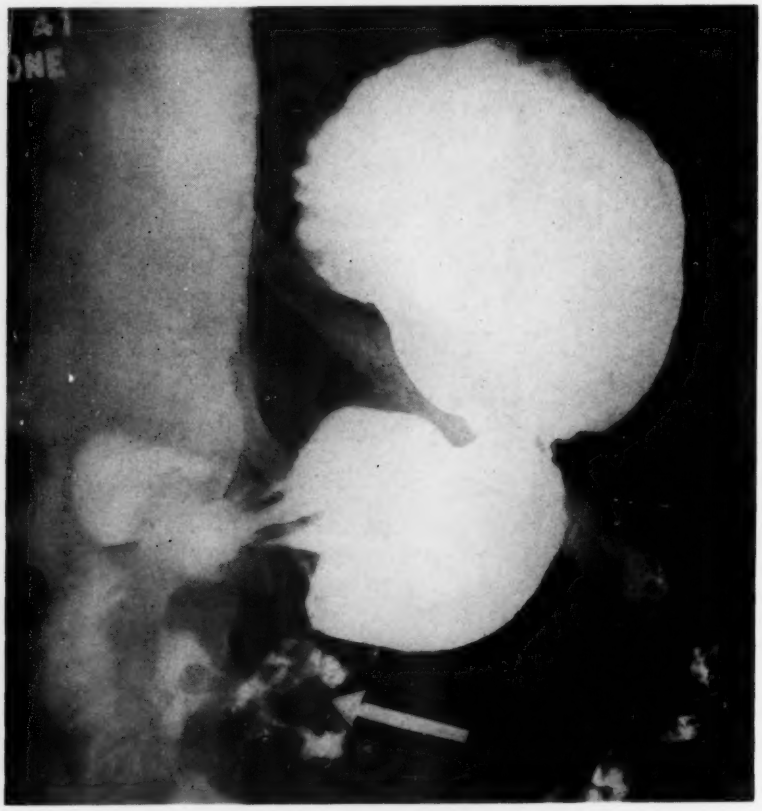

FIG. 4.—Roentgenogram of the stomach and duodenum. Arrow points to the Y-shaped irregularity in distal duodenum, which corresponds to the crater of the actual lesion.

the other two segments. In the suprapapillary group, the onset was acute in about half the cases; the chief symptoms in order of frequency being vomiting, epigastric pain, weakness, weight-loss, jaundice and dyspepsia. Those with gradual onset complained of pain, dyspepsia, weight-loss, vomiting and jaundice in that order. About one-fourth of the cases had a palpable mass in the region of the tumor. The usual roentgenographic picture was that of an ulcerating or obstructing lesion in the duodenum, though in some cases it was negative. The correct preoperative diagnosis was made only twice, and then from the roentgenograms. Roentgenologic studies are chiefly valuable in ruling out lesions of stomach, colon and gallbladder.

The usual pathologic picture is that of cylindrical cell carcinoma, though rare cases of squamous metaplasia have been reported,[32] and a few of malignant adenoma.[21] Hoffman and Pack[21] found that suprapapillary growths were most often of a hard, fibrous or colloid type, rather than scirrhous or polypoid. All three types usually obstruct the lumen and eventually neighboring structures. There is no conclusive evidence that suprapapillary cancer develops on the basis of simple ulcer. The incidence of metastasis is low, due probably to the rapid development of the disease and the early interference with vital functions. Hoffman and Pack[21] report metastasis in 33 per cent of 18 cases, and quote Outerbridge's figure of 20 per cent in 125 cases, though Stewart and Lieber[45] found metastases in 75 per cent of their suprapapillary cases.

Results of treatment are very discouraging. Of course, there is no medical treatment for any duodenal carcinoma beyond palliation and supportive measures. Of 27 cases treated surgically to date, 20 had palliative or purely explorative procedures, with an operative mortality of 13 (65 per cent), while two were reported merely as operative recoveries, four died within the first year, and one at 3½ years after operation. In seven cases in which resections were attempted, three failed to survive operation (42.8 per cent), two are reported merely as operative survivals, one was alive for 3 months and another (Wiig's[51] case) for 7½ months after operation. The operation of choice is resection of the whole duodenal segment, with gastro-enterostomy if the bile and pancreatic ducts are not compromised. If they are, the procedure recommended below for peripapillary tumors is indicated. Palliative procedures are dictated by the pathology; gastro-enterostomy, with or without pyloric exclusion, for relief of alimentary obstruction; cholecystostomy or some type of gallbladder anastamosis for obstructive jaundice.

CARCINOMA OF THE PERIPAPILLARY PORTION

Of all duodenal carcinomata, this group is the most common (65 per cent) and, therefore, the most important. It is impossible to be certain of statistical data in this group because of many contradictions and the confusion of diagnosis in the literature. We have attempted to trace some of these discrepancies to their source and humbly offer the following conclusions, none too sure of their accuracy, since some original sources were not available. Though they themselves were found guilty of several misquotations, the study made by Lieber, Stewart and Lund,[31] in 1939, was found to be the most critical, and we accept their figures as the most accurate available, with some corrections offered.

These authors carefully analyzed 399 cases reported up to 1939, but discarded 194, almost half, for lack of pathologic proof or insufficient data. To 205 acceptable cases, they added 17 new ones, making a series of 222 proven cases to 1939. We have analyzed 45 additional cases to date, of which 29 are acceptable as proven, bringing the current total up to 251.

The new cases are: Fourteen reported by Cooper;[12] six by Hart Hansen;[20] three by Allen,[1] and one each by Cace,[9] Duckworth,[15] Gasbarrini,[18] Hoshino and Abe,[22] Orr,[42] and Cabot,[7] Case No. 24162. Five cases reported from the roentgenographic standpoint only (four by Weintraub and Tuggle,[47] and one by Doub and Jones[14]), were excluded because of insufficient data. Eleven cases, reported surgically, by Whipple,[49] in his excellent article on the surgery of this condition and of carcinoma of the head of the pancreas, have also been excluded from our statistics since no differentiation was made between the duodenal or pancreatic origin of the tumors.

The relatively early development of obstructive jaundice (acute in 80 per cent) is the cardinal symptom of peripapillary carcinoma and was present in 99 per cent of the recorded cases. The jaundice was accompanied by fever in 33 per cent of the cases. The principal accompanying symptoms were pain (60 per cent), loss of weight and strength, anorexia, vomiting, constipation and diarrhea, in that order. Very few patients presented any palpable mass, while 78 per cent had enlarged livers and half the cases had palpable gallbladders. The correct preoperative diagnosis was made in 20 per cent of the cases, and was suspected, roentgenographically, in about 25 per cent. Weintraub and Tuggle[47] reported a case in which the roentgenologic diagnosis was made by the discovery of air in the biliary tree, due to duodenobiliary fistula.

The close proximity in this region of several complex and anatomically variable structures, and the early spread of cancer in the area to adjacent tissues, makes it very difficult to determine the exact site of origin of most peripapillary cancers and to exclude tumors arising in the bile ducts and pancreas. From the standpoint of surgical therapy, such refinements are mainly academic, as the whole area, including the terminal bile and pancreatic ducts and the head of the pancreas may be considered as one unit, but for purposes of nosology an attempt at differentiation is necessary. Lieber, Stewart and Lund[31] classified 229 cases as follows:

1. Primary carcinoma of the ampulla of Vater	3
2. Primary carcinoma of the terminal duct of Wirsung	1
3. Primary carcinoma of the terminal common bile duct	7
4. Primary carcinoma of the intestinal mucous membrane covering the papilla of Vater	3
5. Carcinoma involving all the epithelial structures comprising the papilla of Vater under Groups 1, 2, 3 and 4	182
6. Carcinoma involving all the epithelial structures comprising the papilla of Vater exclusive of the intestinal mucous membrane	33

Thus, 79 per cent of their cases fell into the large, indeterminate Group 5, despite very careful microscopic studies in many instances. Hoffman and Pack[21] believe that "carcinoma of the ampulla of Vater is not a true duodenal carcinoma, since it arises from the epithelium lining the terminal portion of the common bile duct. The growth usually exhibits the histologic characteristics of the epithelium of the bile duct and may even develop true epidermoid carcinoma," of which they present an example. Carcinomata of this region are frequently of the soft, bulky, polypoid variety, with an early

tendency to ulceration and bleeding,[21] which accounts for the fact that about 20 per cent exhibit blood in the stool[31]—a guide to early diagnosis.

From the viewpoint of etiology, Cohen and Colp[11] pointed out that the papilla and ampulla are exposed to the chronic irritation of an alkaline current from the ducts as well as the acid wash of the gastric contents. However, the higher incidence of carcinoma in this region may be due to the fact that the presence in this area of the duct epithelia, which are susceptible to carcinogenesis, outweighs the factor of relative immunity exhibited by the duodenal mucosa itself, for we have seen that it is usually impossible to tell the specific site of origin of most of the tumors.

In evaluating reported surgical results, one is confronted by contradictions in the reports of various authors, based on the loose interpretation of diagnostic criteria for one thing, and also on sheer misquotation in several instances. In 1927, Cohn and Colp[11] reviewed 59 cases treated by radical surgery, reporting an operative mortality of 44 per cent among 53 cases of transduodenal resection. In 1928, Busch[6] reported nine survivals of one year or more, based mainly on Fulde's[17] paper of 1927. Muller and Rademaker,[38] in 1931, quoting Busch,[6] reported eight survivals of four years or more, and are, in turn, quoted by Cooper.[12] In 1935, Hunt and Budd[24] collected 18 additional cases, which added to those of Cohn and Colp, comprised a series of 76 cases subjected to radical surgery with a mortality of 38.1 per cent. Allen,[1] in 1938, reviewed 97 reports of radical surgery (from the above papers) and found a 25.7 per cent survival of one year or more. Basing our present figures mainly on Lieber, Stewart and Lund's[31] critical analysis of the original reports up to 1939, on our own review of the cases reported since then (analyzed by the same criteria), and on reference to original sources where discrepancies appeared among many of the above reports, we have concluded that to date, a total of 136 adequately proven cases of peripapillary carcinoma have been subjected to surgery of any type. In 122 cases, some operative result is known. Of these, 64 had purely palliative procedures for the relief of obstructive jaundice, with an operative mortality of 73.5 per cent, while in 58, radical resection of the primary tumor has been attempted alone or in combination with other procedures, with an operative mortality of 29.3 per cent. Of the latter group, the eight cases below, all of whom had transduodenal

Reported By	Year	Survival Period (Living and Well)	Final Authority for Survival Figure
1. Körte, Case 32	1909	22 years	Busch [6], 1928
2. Oleani	1919	4 years*	Oleani [41], 1919
3. Lewis (Kelly's case)	1921	9 years*	Lewis [29], 1921
4. Kleinschmidt, Case 2	1922	6 years	Lieber, Stewart and Lund [31], 1939
5. Tenani	1922	3 years*	Tenani [46], 1922
6. Fulde	1927	2 years	Fulde [17], 1927
7. Clar	1927	13 years	Nèmenyi, quoted by Lieber, Stewart and Lund [31], 1939
8. Lauwers, Case 1	1933	3 years, 10 mos.	Lieber, Stewart and Lund [31], 1939

* Incorrectly cited by Lieber, Stewart and Lund[31] as Oleani 1 month, Lewis, 4 months, Tenani 5 months.

resection, with or without other procedures, are known to have been living and well for two years or more after operation (13.8 per cent two-year cures), while four of these were alive and well five years or more (6.8 per cent five-year cures)

Also worthy of mention are Muller and Rademaker's[38] case, who died four years and eight months after operation, of metastases, and that of Judd, quoted by Cooper,[12] who was alive 2½ years after operation, but known to have a recurrence, both patients having also had transduodenal resections. Cabot's case, originally reported by Potter[44] as an eight-year cure, was excluded because it was a carcinoma of the common bile duct proximal to the duodenal segment, while three other cases, also reported by Muller and Rademaker[38] and Cooper[12] as five-year cures, were excluded because of insufficient data, namely, the cases of Oehler,[40] Van Remynse and Van Ardenne.†

In the surgical approach to peripapillary carcinoma, the problem of re-establishing biliary and pancreatic continuity, especially the latter, has been most formidable. However, in 1935, Whipple, Parsons and Mullins[50] demonstrated that the reestablishment of pancreatic flow is not essential to life, thus increasing the prospect for a higher percentage of radical cures. The two-stage procedure which they evolved, as revised in 1938 by Whipple,[49] consists of a first stage, in which the common bile duct is ligated, the jejunum is sectioned and a cholecystojejunostomy and entero-anastomosis are performed according to the Y-principle of Roux. At the second stage, gastro-enterostomy is performed, and is followed by a block resection, including the descending portion of the duodenum together with the distal portion of the common duct, and a V-shaped section of the head of the pancreas. The open duodenal ends are closed and the pancreatic ducts are ligated tightly. In 1941, Orr[42] reviewed 15 cases of ampullary and pancreatic carcinoma treated by some variation of Whipple's principle, including one case of his own, and found a 33 per cent mortality, and 26.6 per cent survival (four cases: alive 3 months, 4 months and 6½ months, and one an unknown period, post-operatively, respectively). In the exceptional case of very small tumors, simple excision may be radical enough to get wide of the growth; in others, the two-stage procedure of Whipple appears to be the operation of choice for the future. In very good risks, with adequate preoperative preparation, the two-stage operation is now undertaken in one sitting (Ziegler[52]). Orr[42]

† Oehler[40] originally reported his case in 1910 as alive and well three months after operation. Melchior,[36] in 1917, cited the same case as "alive one year, 11 months post-operative, slightly jaundiced, recurrence? . . . by personal communication of Kausch." This reference was misquoted by Fulde,[17] in 1927, as a six-year cure, the error being perpetuated through Busch[6] (1928) to Muller and Rademaker[38] (1931), and Cooper[12] (1937). The "cures" of Van Remynse (six years) and Van Ardenne (five years) were credited to a report by Klinkert,[26] in 1929, but reference to this source reveals no evidence whatever to consider these cases. They received only the briefest one-sentence mention by a discusser of Klinkert's paper, which reported an entirely different case.

suggests the use of lipocaic (pancreatic hormone) in combination with the above procedure.

CARCINOMA OF THE INFRAPAPILLARY PORTION

This group, in which our own case falls, is the smallest of the three (15 per cent). Stewart, Lieber and Lund[30] analyzed 62 reported cases, accepted 36 as authentic, and added two of their own, making a total of 38 proven cases up to 1937. We have analyzed 19 additional cases, all proven, which, with our case, bring the total known to date to 58. The new cases are: Six reported by Hoffman and Pack,[21] three by Hart Hansen,[20] two by Bergendahl,[2] and one each by Howes,[23] Hadfield-Jones,[19] Allen,[1] Claiborn and Dobbs,[10] Brunschwig and Childs,[5] Pollack,[43] MacIndoe,[33] and Cabot[8] Case No. 27092.

The principal symptoms were pain, vomiting and cachexia, regardless of type of onset, while anorexia, constipation, diarrhea and jaundice were found less commonly. Only 16.6 per cent had a palpable mass in the region of the tumor.[30] The preoperative diagnosis was rarely made. Lieber, Stewart and Lund[30] found in a series of 15 cases studied roentgenographically that an obstructing lesion of the duodenum was present in 40 per cent; in 33 per cent the lesion was incorrectly diagnosed as at or near the pylorus, while in 27 per cent no lesion was demonstrable.

The usual pathologic picture was that of a broad, flat, ulcerating mass, as in our case. The average size was 3–5 cm. in diameter in the majority of cases.

TABLE I

DISTRIBUTION OF CASES AND RESULTS OF SURGICAL THERAPY OF PRIMARY CARCINOMA OF THE DUODENUM

Primary Carcinoma of Duodenum	Suprapapillary	Peripapillary	Infrapapillary	Total
Total No. of reported cases	125	461	84	670
Distribution in duodenum	18.6%	68.8%	12.5%	
Total No. accepted as proved	77	251	58	386
Distribution in duodenum	19.9%	65.0%	15.0%	
Total subjected to surgery	27	136	33	196
Palliative surgery	20	64	22	106
Operability	25.9%	25.4%	37.9%	27.2%
Op. mortality	65.0%	73.5%	90.0%	75.5%
Radical surgery	7	58	11	76
True operability	9.0%	23.1%	18.9%	19.6%
Op. mortality	42.8%	29.3%	33.3%	31.0%
No. alive and well at 2 years	0	8	0	8
Per cent 2-yr. cures	0	13.8%	0	10.5%
No. alive and well at 5 years	0	4	0	4
Per cent 5-yr. cures	0	6.8%	0	5.2%

To date, there are 33 reported cases in which surgical therapy has been applied. Of these, 22 had purely palliative or exploratory operations, in two of which the results are not recorded. Of the 20 known results, 18 were immediate operative mortalities (90 per cent), while all were dead in three months. Eleven radical resections have been attempted, in two of which no results are recorded. Of the nine known results, three were operative mortalities (33 per cent), while six were successful. Of these, three patients

were alive at three months, and three at 15, 16 and 20 months after operation (the cases of Brunschwig and Childs,[5] Hadfield-Jones[19] and Bergendahl,[2] respectively.

Since the bile and pancreatic ducts are not involved, the operation of choice for infrapapillary tumors is resection of the affected segment and reestablishment of alimentary continuity by duodenojejunostomy, end-to-end, end-to-side, or side-to-side. Hadfield-Jones[19] brought the jejunum to the right, under the superior mesenteric artery, until it lay in position for anastamosis to the proximal duodenal segment without tension. Lahey's[28] plan of antecolic duodenojejunostomy, recommended for high jejunal lesions, may also be applied to infrapapillary duodenal lesions.

SUMMARY AND CONCLUSIONS

A proven case of primary carcinoma of the infrapapillary portion of the duodenum is presented, and discussed from the point of view of clinical roentgenographic, operative and autopsy findings.

The literature on all types of primary duodenal carcinoma is reviewed, covering a total of 386 proven cases to date, of which 19.9 per cent were suprapapillary, 65 per cent peripapillary, and 15 per cent were infrapapillary.

Each subgroup is briefly considered from the standpoint of incidence, principal symptomatology, roentgenographic findings, pathologic picture and recorded surgical results. The latter are summarized in Table I.

Earlier diagnosis depends on a better awareness in the mind of the clinician that the diagnosis of duodenal carcinoma is possible though rare (from .03 per cent to .003 of all autopsies). Roentgenologic examination is especially helpful if the lesion is looked for consciously.

While the results of radical surgery in duodena carcinoma so far have been very discouraging (5.2 per cent five-year cures), with earlier diagnosis and with improved methods of pre- and postoperative care, such as exist today, the prospect for improved results in the future looks brighter, since a rational surgical technic is available for each group of cases, and since metastasis at the time of exploration is low.

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SOLITARY NONPARASITIC CYST OF THE LIVER

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Solitary nonparasitic cyst of the liver occurs sufficiently rarely to warrant the report of every such cyst encountered. In 1937, Davis'[11] extensive study of nonparasitic cysts of the liver revealed 187 reported cases of a solitary cyst, to which he added one. Eight years previously, Stoesser and Wangensteen's[38] review placed the number of reported cases at 104. Ochsner[27] reported a case in 1937. Jennings[15] added a case in 1939, and during the same year Attix[2] reported another. In 1940, Montgomery[23] added two cases.

Although reported cases include all ages from fetal life to extreme old age, the disease most commonly presents itself between the ages of 40 to 60. As stated by Montgomery[23] (including his two reported cases) in 1940, there have been only 27 cases under the age of 13. No doubt, as is true in all diseases, there have been others that have not found their way into the literature.

I should like to report a case that presented a solitary nonparasitic cyst of the liver which recently came under my care.

Case Report.—M. S., male, age nine months, was readmitted to the Charlotte Memorial Hospital, September 7, 1941, with an enlarged abdomen since the age of six weeks. The family history was essentially normal. The child was born at full term by normal delivery. The birth weight was eight and one-half pounds (3855.60 Gms.). The child had never eaten well. He nursed only fairly well, and there was frequent regurgitation. He had had no acute sickness other than a "spell of flu" at age two months. The child gained fairly normally up until age six-seven months, since which time he had gained rather poorly. After birth and up until he was four months old, he would never have regular bowel movements, but his diapers were almost constantly spotted. This condition was checked by therapy, but since then there had been some trouble with constipation.

The mother noted that the child's abdomen was too large at age six weeks when "navel bands" were being used. She did not pay a great deal of attention to this, but she became alarmed when she noted a hard lump in the right upper quadrant when he was six months old. Dr. E. K. McLean of the Pediatric Department of the Charlotte Memorial Hospital was consulted. He informed the parents that a tumor was present, and studies were ordered. Dr. Robert W. McKay of the Urological Department studied the kidney status, and it was his opinion that the tumor did not originate from the kidney. Dr. Allan Tuggle of the Radiology Department reported: "A flat film of the abdomen reveals a huge mass in the right abdomen. On some of the films a normal-sized kidney shadow is seen through this mass. The spine is not unusual.

"A barium enema shows the colon to fill completely with no defects or spasms. The mass depresses the hepatic flexure and the midtransverse colon (Fig. 1).

"A pylogram on the following day after injection of five cc. of diodrast shows excretion from each side. A 10, 20, 30, 50, and 70 minute film shows poor function. The mass previously seen on the flat film is excluded as being kidney.

"From the radiographs I doubt that this is a renal neoplasm but would suggest that it is liver."

It was decided to determine whether roentgenotherapy would diminish the size of the mass. Through the anterior right upper quadrant 400 R. and 350 R. through the lateral right upper quadrant failed to decrease the size of the mass.

Physical Examination.—Temperature to be 98°F., pulse 84, respiration 18. The patient was a well-developed and well-nourished white male of nine months, with a

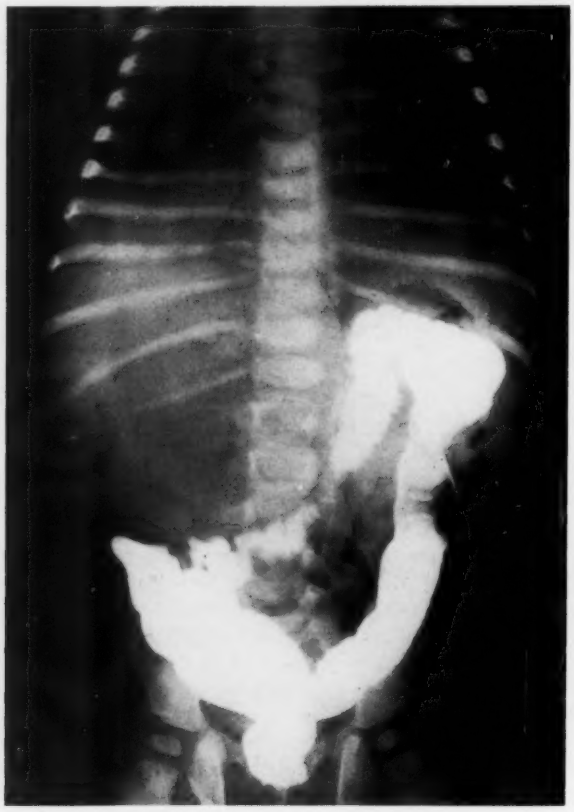

FIG. 1.—Preoperative roentgenogram after barium enema, showing the marked displacement of the colon medially and inferiorly by the large mass occupying the right upper abdomen. As pointed out by Hofmann, this is a most important sign in the diagnosis of liver cysts.

greatly enlarged abdomen. Head: Normal bony contour. Eyes: Reacted actively to light. Ears: Essentially normal. Nose and Throat: Essentially normal. Heart and lungs were negative. Abdomen: There was a large, firm apparently nontender mass about the size of a grapefruit which filled almost the entire right side of the abdomen, especially the right upper quadrant. The mass was fairly movable and seemed to be smooth-walled. There was a visible prominence of the right upper quadrant with a definite flaring of the right costal arch. There were no herniae, scars, or tenderness of the abdomen. Spleen was not palpable. There were no enlarged lymph nodes. External genitalia: normal male development. Extremities were normal.

Laboratory Data.—Hb. 60 per cent, W.B.C. 11,900, R.B.C. 3,900,000, Differential essentially normal; Wassermann and Kahn tests negative. Uranalysis essentially normal.

Impression and Preoperative Diagnosis.—Most likely either a mesenteric or omental cyst.

Operation.—September 7, 1941 (H. S. M., Jr.). The patient was given a subcutaneous infusion of 300 cc. of saline. Under drop-ether anesthesia, the abdomen was opened through a long right midrectus muscle-splitting incision. A large smooth-walled mass, which completely filled the upper right side of the abdomen and a great portion of the right lower abdomen, presented itself. It was immediately apparent that this mass was densely incorporated into the right lobe of the liver. It measured about 12 cm. in diameter, and although firm, it was suggestively cystic. The mass, along with a portion of the liver, was delivered through the incision, which was necessary because it was

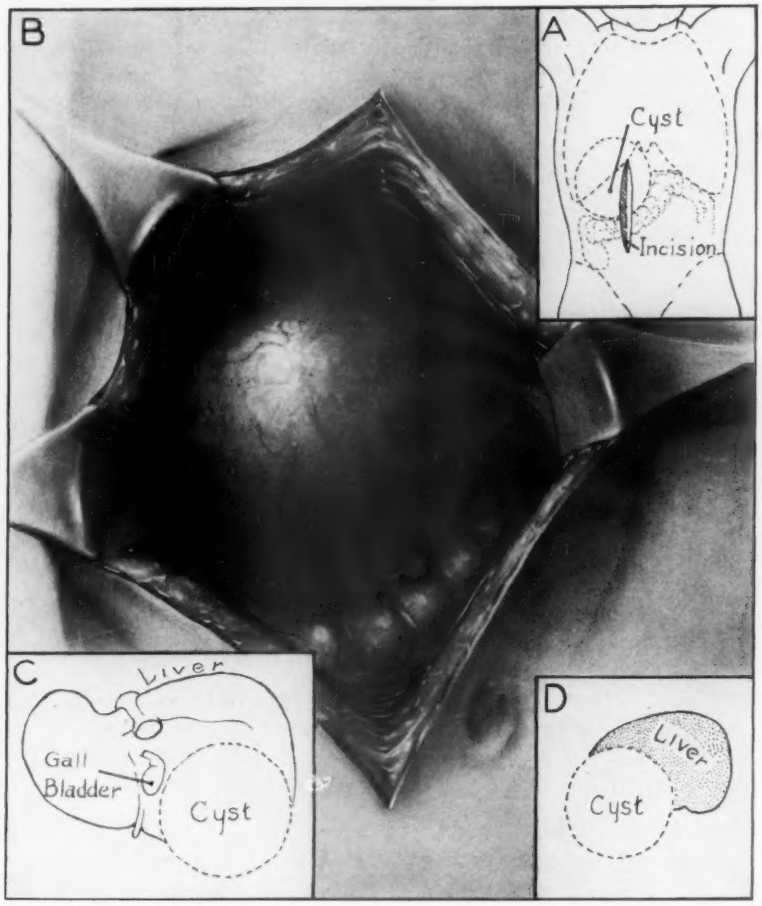

FIG. 2.—Appearance and anatomic relationship of the mass upon operative exposure.

not readily accessible for aspiration. The delivery of the mass and partial delivery of the liver occasioned some shock. The stomach, duodenum, large bowel, and small bowel were all displaced to the left and inferiorly (Fig. 2). An aspirating needle was inserted a distance of fully 2.5-3 cm., when clear straw-colored fluid was encountered and aspirated. After a portion of the fluid had been aspirated, the mass was returned into the abdominal cavity. The gallbladder lay just to the left of the involved portion of the right lobe of the liver. The cyst had taken up fully one-half to two-thirds of the right lobe of the liver. Normal liver tissue could be recognized on the anterosuperior portion

of the cyst, but it was impossible to find a sharp line of demarcation between the grossly normal liver tissue and the wall of the cyst. Abdominal palpation revealed no apparent renal enlargement or pancreatic enlargement that would be suggestive of polycystic disease. The cystic mass was then incised at the point of aspiration. Fluid was removed by suction. Its quantity was not accurately measured although a specimen was saved for study. The interior of the cyst was smooth-walled, and there were several membranous folds partially traversing the cavity. At its most inferior portion the cyst wall was approximately two cm. in thickness. This apparently was the thinnest portion of the wall. An adequate portion of the wall was removed for pathologic study. The cavity of the cyst extended far up into the central portion of the right lobe of the liver. One gained the impression that the cyst most probably had its origin deep within the right lobe of the liver, and that the wall of the cyst was composed of atrophic liver tissue. The intrinsic pressure of the cyst was apparently low. It was apparent that the child would never survive any attempt at complete extirpation of the cyst; hence, marsupialization of the cyst wall to the peritoneum was elected. A portion of the incised cyst was closed with interrupted No. 2 chromic catgut. The peritoneum and posterior rectus sheath were approximated with continuous No. 1 plain catgut. Due to the extreme thickness of the wall of the cyst it was impossible to bring the cyst wall up to the skin margin. The peritoneum and posterior rectus sheath were sutured to the cyst wall about the opening of election in the wall of the cyst. The anterior rectus sheath was approximated with interrupted sutures of fine black silk above and below the opening. Through a large Penrose drain a number of gauze strips that had been saturated with Zenker's solution (solution of bichloride of mercury 5 per cent, and potassium dichromate 2.5 per cent) were inserted into the cyst cavity. The patient left the operating room in fair condition. *Postoperative Diagnosis:* Solitary nonparasitic cyst of the liver.

Pathologic Examination.—Microscopic: Dr. Paul Kimmelstiel: "Histologic sections through the wall of the cyst show that the latter is mainly composed of rather a cellular connective tissue in which are found numerous bizarre-shaped ducts lined by a single layer of low cuboidal epithelial cells. The lumen of many of the ducts is open, but some of them are not patent. The ducts and capillaries are irregularly arranged and surrounded by concentric layers of laminated connective tissue. In some areas a collection of large polygonal epithelial cells is seen; the cells have clear borders and the cytoplasm is vacuolized. The nucleus is located in the center. The cells are not in distinct arrangement; they do not form glands, but they occur in columns and nests, separated from each other by capillaries which can be readily identified as they are lined by endothelial cells. These groups of cells are suggestive of vacuolized liver cells which do not bear cilia. The inner lining of the cyst is in continuity with a number of bay-like extensions which in turn are continuous with the above described ducts in the wall of the cyst.

"Chemical examination of fluid from cyst: Specific gravity 1.010, N. P. N. 13 mg. per cent, cholesterol very faint trace, bile negative, urobilinogen negative, hemoglobin negative, total protein 1.5 Gm. per cent. *Pathologic Diagnosis.*—Congenital cyst of liver derived from bile ducts."[11]

Postoperative Progress.—Following operation there was a febrile reaction, the rectal temperature rising to 104.4°F. on the evening of the day of operation, but this gradually subsided. The gauze pack was removed from the liver cyst on the second postoperative day, and a soft rubber catheter was inserted. Drainage was only moderate. At no time was there any gross or clinical evidence of bile. On the ninth postoperative day, the temperature suddenly rose to 104°F., but rapidly subsided with sulfathiazole therapy. The cyst was daily irrigated with Dakin's solution. Frequent measurements demonstrated gradual diminution in the size of the cyst. One month after operation a roentgenogram taken following injection of lipiodol through the catheter showed a cavity of about two cm. in size (Fig. 4). The liver shadow was still large. The

cyst was frequently flooded with Zenker's solution which seemed to produce little or no generalized reaction.

Subsequent Course.—The child was discharged from the hospital, October 15, 1941, five weeks after operation, and was seen at weekly intervals, the catheter being irrigated with Zenker's solution without any untoward reaction at each visit. The cavity gradually reduced to one cm. in size. The catheter was gradually removed and the tract was packed with iodoform gauze. This gauze was gradually displaced by granulation tissue. Abdominal palpation, March 30, 1942, revealed an oblong, nontender, firm mass that extended from the area of marsupialization to the liver. This was taken to be the obliterated cyst of the liver. There was a small granulating area at the region of the

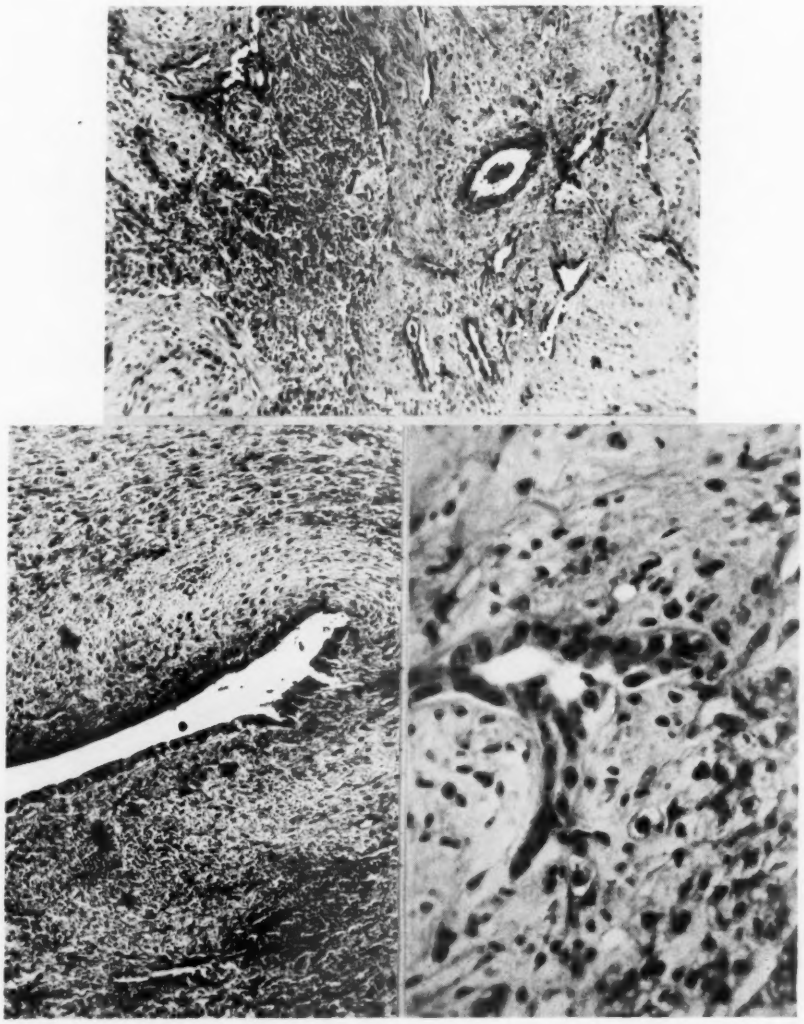

FIG. 3.—Top: Cyst wall under low power showing fibrous tissue with bile ducts. On the left of the midline is nest of liver cells.
Lower right: Outpouching of cyst, showing high cylindrical cells lining the inner wall.
Lower left: Cyst wall under high power showing small bile ducts partially with and partially without demonstrable lumen.

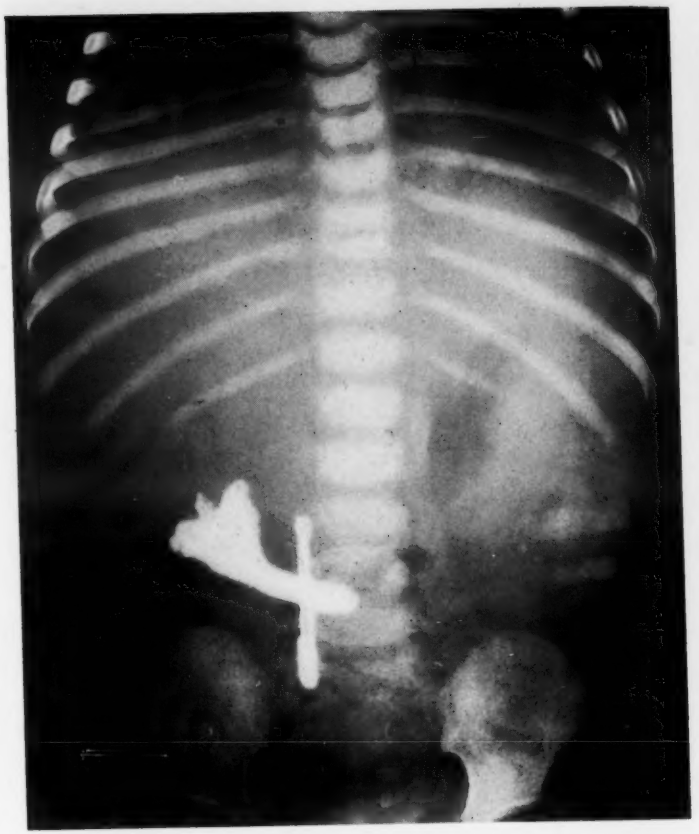

FIG. 4.—Postoperative roentgenogram, October 8, 1941, following injection of lipiodol through a catheter placed at the point of marsupialization.

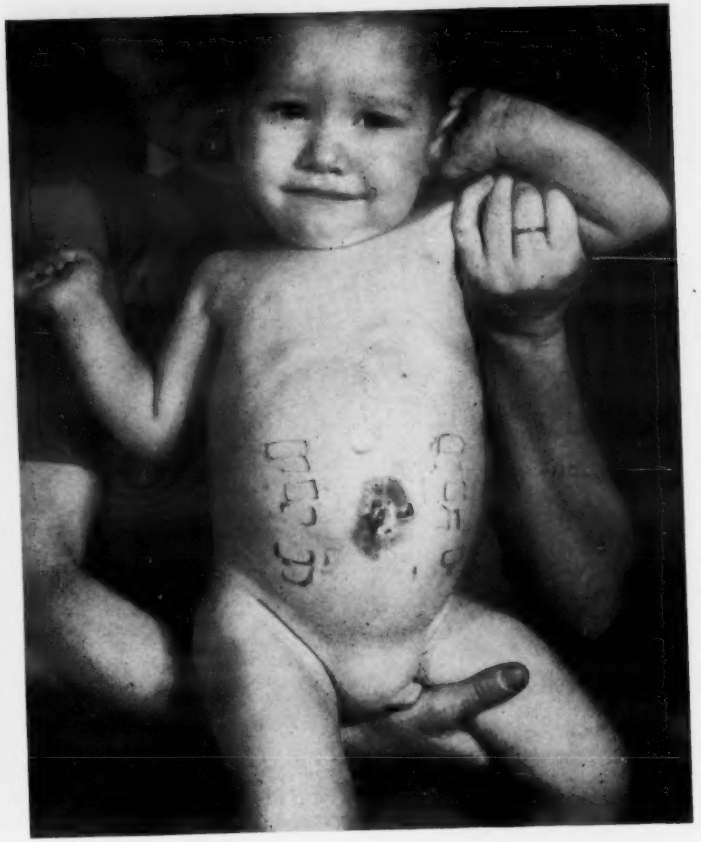

FIG. 5.—Photograph taken, March 24, 1942. The upper abdomen seems rather full. The mass can be palpated, but is considerably reduced in size. The sinus tract is closed, and there is no drainage.

marsupialization that is rapidly healing under treatment with silver nitrate. The sinus tract was obliterated. The child is growing and developing normally, and appears to be in the best of health (Fig. 5).

Nonparasitic cysts of the liver may be single or multiple. Multiple cysts may be confined to the liver alone, but more commonly they are associated with cysts of the kidneys, pancreas, spleen, lungs, and brain. Multiple cysts are more common in children than in adults, and may be associated with other congenital malformations as harelip, cleft palate, and spina bifida. Solitary nonparasitic cysts are more common in adults. Whether justifiable or not, it is customary to differentiate sharply between solitary and multiple nonparasitic cysts of the liver. Boyd [8] and Sonntag [36] are inclined to believe that cystic disease and solitary cysts of the liver are different manifestations of the same disease. Stoesser and Wangensteen [38] state as follows: "It is generally accepted that solitary cysts in nonarteriosclerotic kidneys are but another expression of polycystic disease; a corresponding similarity would appear to extend to the manifestation of the disease in the liver. In several cases reported on solitary cysts of the liver, there have been small cysts in the proximity of the large cyst." Whether the solitary cyst of the liver is a lonesome manifestation of polycystic disease or an entirely unrelated abnormal process is a debatable issue. Until more is known, and for the sake of clarity, let us refer to these two conditions as definitely separate entities. In the reports of some writers this differentiation has not been sharply adhered to.

Cysts seem to occur more often in females than in males, the ratio being 4:1. This is explained on the basis that developmental defects are well known to be more common in the female sex.

The earliest reported case of cystic disease of the liver was that of Bristowe in 1856. This was the postmortem finding in a 53-year-old shoemaker. To substantiate the congenital origin of these cysts and to illustrate the age variations, let us review some of the reports: In 1880, Witzel [44] reported a case of a large liver cyst in a newborn which complicated labor. In the same year Sanger and Klopp [32] reported a very similar case. In 1892, Bagot [3] reported a case with a liver cyst containing two and one-half gallons of fluid which, of course, interfered with delivery. The oldest patient with nonparasitic cyst of the liver was reported by Bland-Sutton[6] in 1905. This was in a man, age 75.

PATHOLOGIC ANATOMY

Reference has already been made regarding solitary and multiple nonparasitic cysts of the liver. Solitary cysts are most commonly found at the antero-inferior portion of the right lobe of the liver. The quadrate lobe and the left lobe are sometimes involved, and in still fewer cases, the cyst occupied the central portion of the liver. In one case described by Montgomery,[23] and in a few cases described by Harrington,[13] the round ligament was involved. The size of the cyst and the amount of liver destruction vary considerably. Some cysts are microscopic, and some may fill the abdomen. An

entire lobe of the liver may be destroyed by the cyst. The cyst may be wholly or partially intrahepatic, or it may be pedunculated. Little is known about the growth of these cysts, but apparently growth is so tardy that adjacent viscera quickly adapt themselves to its presence without symptoms as a rule. In contradistinction to echinococcus cysts, these cysts have a characteristic low internal tension.

The external surface of these cysts is usually smooth, glistening, and grayish-blue, often showing many dilated veins. The appearance may be not unlike that of a large smooth-walled cyst of the ovary. The internal surface is usually smooth, less regular, and is usually trabeculated. The thickness of the cyst wall is variable. There is usually no definite line of cleavage between the intrahepatic and partially intrahepatic cyst wall and the normal liver tissue.

The cystic contents vary from a clear, watery, sometimes yellowish-brown fluid, neutral or alkaline in reaction, with a specific gravity varying from 1.007 to 1.024 to a semisolid material resembling an organized clot, as in the case reported by Montgomery.[23] An analysis of the cystic fluid may show albumin, mucin, cholesterol, blood, hematoidin, hemosederin, tyrosine, some granular and cellular débris, and rarely bile. Some writers claim that the older cysts contain no bile because it is being constantly absorbed. In explanation of the presence of bile in some cysts, Boyd[8] ventured to say that if the cyst increases in size, large intrahepatic bile ducts rupture into the cyst, their walls being destroyed by pressure. This explanation would appear to find support in the fact that following drainage of the cyst, bile has been known to escape through the drainage tube when the contents of the cyst at operation failed to show bile pigments.

The microscopic appearance of the cyst wall, according to various authorities, consists of three layers of connective tissue, an inner layer rich in cellular element, a circularly arranged dense layer poor in cell nuclei and containing a few blood vessels with thickened intima, a loose outer layer with elastic fibers, muscle bundles rich in cells, many blood vessels and bile ducts. The cyst is lined with cubical, cylindrical, or flat cells, but the epithelium is much desquamated. The bile ducts found in the walls of the cysts are usually lined with the same kind of epithelium which lines the cyst.

The most generally accepted classification of nonparasitic cysts of the liver is that of Sonntag:[36] "(a) Blood and degenerated cysts; (b) dermoid cysts; (c) lymphatic cysts due to obstruction or to congenital dilatation of the lymphatics; (d) endothelial cysts; (e) cysts due to bile duct obstruction; and (f) proliferative cysts (cystadenoma)."

The possible mode of origin of these cysts has been discussed at length by Siegmund,[35] von Kahlden,[40] Neuwerk and Hufschmid,[26] Borrmann,[7] Kaufman,[17] Konjetzny,[18] Sternberg,[37] Plenk,[31] Burns,[9] Jones,[16] Bland-Sutton,[6] Moschowitz,[24] McGlannon[22] and Henke.[14]

There is considerable discrepancy in regard to the histiogenesis depending on the individual interpretation of histologic findings. Consequently, no classification of nonparasitic cysts of the liver is yet presented which has

found general acceptance. Herxheimer,[14] in his description of cystadenoma of the liver (cysts), correctly emphasizes this point.

DIAGNOSIS AND CLINICAL FEATURES

Solitary nonparasitic cysts of the liver apparently grow so slowly that in most instances, few if any symptoms are produced, advice usually being sought because of the presence of a painless abdominal swelling. Occasionally, however, there may be a sudden onset of acute symptoms. This is seen, for instance, when an acute hemorrhage occurs into the cyst, when suppuration supervenes, or when, in the pedunculated variety, the pedicle becomes twisted. A correct preoperative diagnosis is rarely made, the majority of the cases being diagnosed on exploratory celiotomy or at necropsy. There is only one case reported with a correct preoperative diagnosis. Kilvington, of Melbourne (where hydatid cysts are common), made this diagnosis on the basis of the low internal tension of the cyst.

This condition is usually symptomless unless complications occur or the cyst compresses some adjacent structure such as the extrahepatic bile ducts or the first portion of the duodenum. With compression, there may be acute hepatic pain, nausea, vomiting, or jaundice. As most of these cysts arise from the antero-inferior surface of the right lobe of the liver, clinical examination will determine the site of the tumor while radiography may show that the tumor is part of the liver. One of the most important signs is that pointed out by Hofmann. With large cysts or tumors of the liver, the colon is pushed downward and to the left of the abdomen, while in the case of neoplasm or other enlargements of the kidney, the colon lies over the tumor. This point may be easily demonstrated by barium enema. In my opinion, this a very important diagnostic criterion.

In the case of all large pedunculated cysts of the liver, downward displacement is limited by anchorage to the liver, but a considerable degree of lateral movement is often obtained. Induced pneumoperitoneum followed by roentgenologic examination has proven of little help. Peritoneoscopy may be an aid in diagnosis. Liver function tests, as a rule, show little deviation from the normal. I do not intend to enter into a detailed discussion of the differential diagnoses at this time.

TREATMENT

The only treatment for solitary cysts of the liver demanding relief is surgical intervention. Complete extirpation of the cyst should be undertaken if conditions permit. Too frequently, this procedure cannot be wisely attempted without inviting disaster. Partial extirpation along with drainage and marsupialization, or just drainage along with marsupialization have produced satisfactory end-results in most cases. In some instances in which external drainage has been done, a sinus has remained for from a few months to years. In Evans' case a sinus persisted. Jones [16] stated that in Theodoroff's

case a sinus remained for 37 months following drainage of a nonparasitic cyst. Porter's patient developed a chronic sinus, and the patient died following an attempt to correct this condition surgically. Fatalities have been reported which were due to shock from the sudden release of intra-abdominal pressure. This can be avoided by the injection of epinephrine hydrochloride subcutaneously before the cyst is opened, and by slow removal of the fluid. The prognosis is good if the patient survives operation, but the operative mortality of nonparasitic cyst of the liver in reported cases varies between 10 and 30 per cent.

COMMENT.—In my case, I should like to stress the extreme thickness of the cyst wall, and the fact that a large portion of the cyst was intrahepatic. Although the fluid content of the cyst was aspirated by needle, the cyst showed no tendency to collapse. The thickness and rigidity of the wall would not have permitted any radical removal of the cyst, nor would an adequate closure have been possible after such a procedure; furthermore, this patient would not have survived such a radical attempt at removal of the cyst. These considerations prompted the operative procedure adopted. A portion of the wall was excised for pathologic study, and the cyst wall was marsupialized to the peritoneum and the posterior rectus sheath. No collapse of the thick wall of the cyst was anticipated. The lining was frequently treated with Zenker's solution to facilitate the eventual obliteration of the cavity by granulation tissue. The end-result is quite satisfactory.

Our case, as well as the majority of similarly treated ones, may throw some light upon the nature of such nonparasitic cysts of the liver which are lined by epithelial cells. It is generally agreed that these cysts, and they represent the majority of cases, are derived from congenital malformations. The mode of growth, however, has not satisfactorily been explained.

With the exception of such few cases, in which histologic examination revealed evidence of the carcinomatous character of a papillary cystadenoma, the true blastomatous nature of liver cysts is still questionable.

They can readily be classified as hamartomata in the sense of Albrecht, as malformations based upon disproportion anlage of tissue elements.

The enlargement may be explained by gradual increase of fluid with proportionate increase in number of lining epithelial cells within physiologic limits. Adjacent smaller cysts may merge into the larger one. Pressure upon the surrounding liver tissue may result in atrophy. The latter, as commonly observed in cirrhosis or other scars of liver tissue, is associated with proliferation of bile ducts. Proliferating bile ducts, therefore, in the wall of liver cysts, as observed in our and other cases, does not necessarily indicate the true blastomatous nature of the cyst. Secondly, they may communicate with the main lumen but can readily be interpreted as the result rather than the origin of the cyst.

This interpretation, in my opinion, seems likely because our case, as well as other similarly treated cases, have shown an obliteration and complete cure following destruction of the inner epithelial lining. If the tortuous bile

ducts deep in the thick fibrous wall of the cyst would truly be blastomatous in nature, the majority of cysts would have shown recurrence.

It is for this reason that I believe that epithelial-lined cysts, which fail to show active papillary growth, should be classified as hamartomata rather than cystadenomata in the sense of true blastomata.

SUMMARY

A solitary nonparasitic cyst of the liver in a nine months old child is presented. Because of the impossibility of complete extirpation, drainage and marsupialization was done. The end-result is very satisfactory, a complete cure being anticipated. Reasons are presented for a classification of this liver cyst as hamartoma rather than cystadenomatous blastoma.

Grateful acknowledgment is made of the help of Dr. Paul Kimmelstiel, of the Department of Pathology, Dr. Allan Tuggle, of the Radiology Department, Dr. Robert W. McKay, of the Urological Department, and Dr. E. K. McLean, of the Pediatric Department of the Charlotte Memorial Hospital, Charlotte, N. C.

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STUDIES ON THE USE OF METALS IN SURGERY*
PART II
EXPERIMENTS ON THE USE OF TICONIUM IN CRANIAL REPAIR

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IN A PREVIOUS COMMUNICATION[1] we have reported assays of cytotoxicity of certain metals in tissue cultures of chick embryo fibroblasts. Our purpose in that study, and in the present one, has been to find an alloy that conforms to the requirements of bone surgery and, hence, for the repair of cranial defects.

As was pointed out in that paper, an ideal cranioplastic substance should be nontoxic, strong and, at the same time, light enough to keep the bulk of the appliance small. In addition, it should be so malleable as to permit working and shaping during the operation. This latter characteristic is an exceedingly important one in cranial repair because casting is both time-consuming and expensive. For a cast plate to fit perfectly it is necessary to secure an impression of the defect itself. This is a cumbersome and often undesirable procedure to attempt at the time of operation. Many times the bone flap serves as a poor pattern either because it is diseased or the use of the rongeur has changed the shape of the defect after its removal. Estimation of the required plate by roentgenograms and measurement through the intact scalp is difficult. Finally, a cast plate, if found not to fit well, cannot easily be altered at the operating table.

The casting alloy Vitallium has been thoroughly studied by Venable, Stuck, and Beach.[2, 3, 4] It is light, strong and nontoxic. It has been used for cranial repair by Geib,[5] Peyton and Hall,[6] and by Beck.[7] Attention has been called to its lack of malleability as being undesirable. Its use in bone work in general, however, has been so satisfactory that we have used it as a control substance in our experiments, despite the fact that it does not completely fulfill the peculiar requirements for cranial repair.

The wrought alloy Ticonium is light and strong and, in addition, malleable. In the form of thin, perforated plates it can be cut with tin shears and molded by hand or with pliers. The perforations permit the making of slots or wedges to allow for unusual shapes, and provide many choices for screw holes.

Our previous study showed that wrought Ticonium and Vitallium and cast Ticonium (with beryllium), were not toxic to chick fibroblasts in tissue culture. The present paper reports results with these three alloys in repair of cranial defects in dogs. Some observations on electrolysis have been added.

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* Read at the Annual Meeting of the Harvey Cushing Society in New York, N. Y., May 21, 1942.

OBSERVATIONS ON DOGS

Method.—Three different alloys were used: Ticonium "wrought" (nickel 36.2%, cobalt 29.6%, chromium 28.2%, molybdenum 6%); Vitallium (cobalt 65%, chromium 30%, molybdenum 5%, manganese, silicon); and Ticonium "cast" (nickel 35.6%, cobalt 29.1%, chromium 27.7%, molybdenum 6%, beryllium 1.6%)

In Group I we used material prepared with the wrought alloy Ticonium* only. This material consisted of sheets cold-rolled, with 30 per cent cold

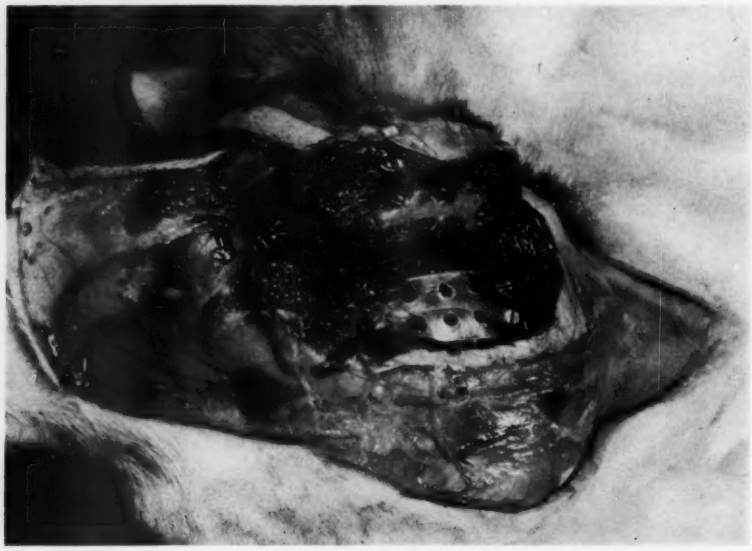

FIG. 1.—(Dog 52)—"Wrought" Ticonium: Plate over the temporoparietal region. Eleven months after operation, showing the absence of corrosion of the metal. Muscle overlying the plate and growing through the perforations has been reflected.

reduction subsequent to the final anneal. This seemed to be preferable for experimental purpose. The plates had a thickness of 0.17 cm. (27-gauge U. S. Standard). The sheets had been punched at intervals of 0.4 cm. with holes 0.3 cm. in diameter. They were then polished, thus taking the punch marks out (Fig 1). Prepared in this manner, the plates were easily cut and molded at operation.

The plates used in Groups II and III were cast in varying size and thickness, according to the shape of dogs' skulls. Each plate had three attachments for the screws. In most of the plates, four to five holes were punched. A few solid plates were used. In Group II, machine made screws of Vitallium,† and in Group III hand made screws of Ticonium "cast," were used.

* The material made of Ticonium ("wrought" and "cast") was prepared by the Research Laboratories of the "Ticonium" Co., Albany, N. Y.

† The Vitallium plates and screws were furnished by the Austenal Laboratories, New York, N. Y.

Technic: Healthy, well-nourished mongrel dogs of various ages were selected. The dogs were kept in separate cages and well fed. The animals were anesthetized by intraperitoneal injection of nembutal (Abbott) (Max. dose 0.7 cc. of a 5 per cent solution per Kg. body weight). The entire head or thigh was shaved and cleaned with a brush, soap, and water for a period of five minutes. Using strict aseptic technic, a unilateral U-shaped skin flap was turned down over the frontotemporoparietal region. The "temporal" muscle was sharply dissected from the underlying bone. The periosteum was scraped off, and, in the center of the denuded area of the bone, an opening was made by use of a trephine. This opening was enlarged to an average size of 2 x 3 cm. Throughout the procedure hemostasis was secured by use of the cautery. The dura remained closed with the exception of those experiments calling for the insertion of metal pieces into the cortex. In these instances a nick was made into the dura, the cortex was incised and a piece of metal about 0.2 x 0.3 cm. was inserted vertically into the cerebrum.

In Group I a wrought Ticonium screen was used, having been previously autoclaved. From such a screen a plate was cut with tin shears to overlap the defect at all four margins by about 0.3 cm. This plate was then molded by hand to the convexity of the skull, and in each instance very satisfactory plastic repair was obtained. The plate was then fastened to the bone with 2-4 screws drilled into the bone to full length. Closure was performed in layers with the exclusive use of interrupted sutures of fine black silk. Skin closure was obtained with interrupted subcuticular sutures of the same material. A bandage dressing was applied and secured by an encasement of plaster of paris around the head and neck. The encasement remained in place for about five days.

Cast plates were used in the experiments of Group II and III. These plates, after having been autoclaved, were placed over the defect and fastened by screws drilled into the bone surrounding the defect.

Operations upon the femur were performed under the same aseptic conditions. A longitudinal incision was made over the lateral surface of the thigh. The muscles were bluntly dissected and retracted. The periosteum was removed, screw holes were drilled into the bone and the screws inserted. Closure was done in layers as above.

During the first postoperative day, fluids in the form of normal saline solution were administered subcutaneously. On the second postoperative day the dogs resumed their usual feeding. Body temperature was determined during the first postoperative days, and the dogs were reexamined at regular intervals. Roentgenograms were taken of a limited number of animals.

The dogs were sacrificed by intracardiac injection of ether or nembutal. Autopsy was performed, and the tissue fixed in 4 per cent neutral formaldehyde solution. Blocks were selected from the bone, and any grossly diseased viscera noted. These were embedded in paraffin or celloidin, and sectioned. Sections were studied with one or more of the following stains: Hematoxylin and eosin, Mallory's phosphotungstic acid hematoxylin, Masson's trichrome (Goldner's modification) and Giemsa.

GROUP I: TICONIUM "WROUGHT"

Experimental Results.—Twenty-one dogs were used for the experiments in this group. The tissues were exposed in all to a total of 115 pieces of metal: Fourteen plates, 78 screws in the skull bone, 19 screws in the femur and four pieces of metal which were embedded into the cerebral cortex. The dogs were observed from two days to 14 months; 17 animals were observed for a period of longer than 10 months. Fourteen dogs were sacrificed, and seven dogs died on account of secondary illness.

CASE REPORTS

Dog. 25.—*Operation:* July 11, 1940. Plate secured with 2 screws. 7th p.o. day dog injured flap by scratching; secondary subcutaneous infection. Healed in 3 weeks. Sacrificed August 6, 1941.

Macroscopic: Scar at operative site not unusual. Screws firmly embedded. Plate well attached, enclosed in a fibrous bursa-like sac. Metal shiny. No fluid. Bone defect filled by firm fibrous tissue

to outer surface of which muscle is attached and grows through perforations in the plate. Dura merges with this fibrous layer. Underlying cortex normal.

Microscopic: Plate bed composed of smooth layer of laminated, partially hyalinized fibrous tissue. Rare macrophage in looser connective tissue especially about blood vessels. Some marginal new bone formation.

Dog 36.—*Operation:* July 13, 1940. Plate secured with 4 screws. Flap healed *per primam.* 6th p.o. day semistuporous; drainage from eyes and nasal mucous membranes. Slight motor weakness of both hind legs. "Distemper." Died July 21, 1940.

Macroscopic: Operative site healing well. Small hematoma in superficial fascial layer. Surface of muscle overlying plate grayish-brown. Small amount of cloudy reddish fluid immediately about plate. Screws secure. Plate firmly attached. Metal bright and shiny. Dura underlying plate intact but dark red. Not adherent to cortex. Right side of heart dilated. Lungs edematous. Left kidney hydronephrotic.

Microscopic: Focal edema of lungs. Chronic pyelonephritis. Hyperemia of liver. Cranial muscle focus of organizing fibrinous exudate and regenerating muscle. Bone margin shows organizing fibrinous mat.

Dog 38.—*Operation:* July 23, 1940. Metal inserted into cortex. Plate secured with 4 screws. Flap healed *per primam.* Sacrificed September 4, 1941.

Macroscopic: Scar of operative site not unusual. Screws firmly embedded. Plate securely enclosed in bursa-like sac. Metal bright and shiny. No fluid. Bone defect bridged by firm fibrous layer, merging with dura. Cerebral cortex surface adherent to dura over an embedded piece of bright, shiny metal which is surrounded by a thin white capsule.

Microscopic: Plate bed consists of laminated fibrous tissue. At bone margin is considerable new bone almost bridging the gap. Muscle fibers are oriented perpendicular to the plate bed. No inflammation or necrosis. Rare macrophage about blood vessels of muscle.

Dog 40.—*Operation:* July 25, 1940. Plate secured with 4 screws. Following day: Fever, tachypnea, sputum (mixed bacteriae). Sulfapyridine. Died after 30 hours.

Macroscopic: Generalized adiposity. Skin flap flat and soft. Fascia sutured tightly. Plate firmly secured by 4 tightly embedded screws. Metal smooth and shiny. Between plate and dura thin layer of clotted blood. Dura smooth and not adherent. Dilatation of right side of heart. Large foci of consolidation in lungs. Uterus contains 2 small fetuses.

Microscopic: Obturation of bronchi by mucous and aspirated foreign material. Massive atelectasis. Thin layer of erythrocytes and fibrin above dura. Small fibrinous clot in screw hole. Muscle edematous, infiltrated with neutrophiles. (Period of observation too short. Findings in accord with wound healing at this stage.)

Dog 41.—*Operation:* July 26, 1940. Three screws inserted into frontoparietal region. Three screws inserted into femur. Scalp flap healed *per primam.* Lower third of leg incision healed secondarily 15th p.o. day. Sacrificed September, 1941.

Macroscopic: Operative sites show usual scarring. Screws firmly embedded. Metal shiny.

Microscopic: Skull screw hole lined by thin fibrous membrane immediately beneath which are rare macrophages containing pigment. Loose fibrous tissue covers head. Some laminated new bone beneath part of tract lining. Femur screw hole essentially similar.

Dog 42.—*Operation:* July 27, 1940. Three screws inserted into frontoparietal bone. Flap healed *per primam.* 12th p.o. day: Fever, tachypnea. Died August 13, 1940.

Macroscopic: Operative wound well healed. Screws firmly embedded, have penetrated inner table. Metal shiny. No fluid. Dura quite vascular. Underlying cortex negative. Dilatation of right chambers of heart. Massive consolidation of lungs.

Microscopic: Necrotizing bronchopneumonia. Screw holes, thin fibrous tissue lining with a few lymphocytes in vicinity. Bone undergoing remodelling.

Dog 43.—*Operation:* July 30, 1940. Metal inserted into cortex. Plate secured with 4 screws. Flap healed *per primam.* Sacrificed August, 1941.

Macroscopic: Scar of operative site not unusual. Plate firmly embedded in bursa-like sac. Screws secure. Metal smooth and shiny. Muscle has grown through perforations in plate. Firm fibrous tissue bridges the bony defect and merges with the dura. No adhesions to brain. Piece of bright metal securely embedded in cerebral cortex and invested by a thin white capsule.

Microscopic: Brain: Metal enclosed in a sac, inner lining of which is hyalinized collagen. This is separated from brain by a thin, clear space containing a few macrophages several of which enclose black iron-containing pigment. The space is bounded externally by a poorly defined thin layer of disorderly gliosis. Skull: Screw holes and plate bed show usual fibrous tissue lining without inflammation or granulation tissue. Rare pigmented macrophages in connective tissue especially about blood vessels.

Dog 44.—*Operation:* July 31, 1940. Dura thickened and roughened. Plate secured with 4 screws. Flap healed *per primam.* For 4 weeks dog continued to do well. Exact data as to further course not available. September 12th, dog was found dead.

Macroscopic: Animal emaciated, vomitus in mouth. Scar of operative site not remarkable. Plate firmly embedded in bursa-like sac. Metal smooth and shiny. No fluid. Bony defect filled by firm

fibrous tissue. Small piece of shiny metal attached to dura by few adhesions. Cortical surface not adherent. Cecal diverticulum adherent and acutely inflamed.

Microscopic: Plate bed lining thin fibrous tissue layer. No necrosis or inflammation.

Dog 45. *Operation:* August 2, 1940. Plate secured with 3 screws. Flap healed without infection. Dog did well until latter part of November: Diarrhea, vomiting. Died November 29, 1940.

Macroscopic: Plate and screws shiny. Plate embedded in bursa-like sac. Bone defect filled by fibrous tissue. Underlying brain negative. Entire sigmoid edematous, hyperemic and ulcerated. No peritonitis.

Microscopic: Colon: Acute colitis with mucosal ulceration. Skull: Plate bed thin fibrous layer in which is some new bone formation partially bridging the defect.

Dog 46.—*Operation:* August 2, 1940. Plate secured with 4 screws. 3rd p.o. day papilledema on the right. Disappeared after one week. Skin flap healed *per primam.* October 18, 1940, litter of eight puppies. Low grade fever for 2 months, evidence of fatigue and exhaustion. Impression: "Low grade infection." Recovered by January, 1941. Clinically, remained well until sacrificed September 23, 1941.

Macroscopic: Skin and fascia at operative site appeared negative. Muscle overlying the plate shaggy, soft, necrotic. Cystic space between muscle and metal contained 4 cc. of yellowish-red serous fluid. The usual fibrous sac about metal incomplete. Metal smooth and shiny. Three or 4 screws loose. Bone defect replaced by firm fibrous layer with a rough surface. Underlying cortex not adherent, grossly negative.

Microscopic: Brain: Essentially negative. Skull: Plate bed composed of a thick layer of chronically inflamed granulation tissue in the inner layer of which are many neutrophils and eosinophils; acidophile necrotic masses are present in places along the lining. The bony defect is bridged by dense fibrous tissue containing foci of new bone formation. In this fibrous bridge are a number of small compact aggregates of lymphocytes and pigment bearing macrophages. Bacterial stains reveal no organism.

Dog 48.—*Operation:* August 7, 1940. Plate secured by 4 screws. Flap healed *per primam.* 20th p.o. day drainage from eyes and nasal mucous membranes. Moist râles, right lung. Despite sulfapyridine, progressive cachexia, loss of appetite, general weakness, fibrillary twitchings of muscles. "Distemper." Died September 8, 1940.

Macroscopic: Plate and screws firmly attached. Shiny and smooth. Underlying dura merges with fibrous tissue layer, filling bone defect. Focal pneumonia consolidation of lungs.

Microscopic: Lung: Necrotizing pneumonia. Skull: Plate bed and screw hole lined by thin compact fibrous tissue layer. Few pigmented macrophages.

Dog 51.—*Operation:* August 6, 1940. Plate secured by 4 screws. Post. med. screw not tight. Post. lat. screw inserted obliquely. Flap healed *per primam.* 15th p.o. day diarrhea, progressive cachexia. No clinical pulmonary findings. Died August 27, 1940.

Macroscopic: Operative site insignificant. Plate firmly embedded. Screws secure except for post. med. screw which appears movable but in place. Firm fibrous tissue bridges the defect and merges with the dura. Underlying cortex not unusual. Diffuse pneumonitis. Diffuse purulent bronchitis, with sparing of lower lobe of left lung.

Microscopic: Skull: Plate bed shows no sign of inflammation or necrosis.

Dog 52.—*Operation:* August 8, 1940. Plate secured with 4 screws. Post. lat. screw inserted obliquely. Flap healed *per primam.* Sacrificed September, 1941.

Macroscopic: Plate securely embedded in bursa-like sac. Screws firm. Metal smooth and shiny. Firm fibrous tissue bridges the defect in the bone and merges with the dura. Underlying cortex not unusual.

Microscopic: Fibrous plate bed. No inflammation or necrosis.

Dog 55.—*Operation:* August 14, 1940. Plate secured by 4 screws. Flap healed *per primam.* Sacrificed September, 1941.

Macroscopic: Plate firmly embedded in bursa-like sac. Screws tight. Metal smooth and shiny. The bony defect is bridged by firm fibrous tissue which merges with the dura. The underlying cortex does not show any gross changes.

Microscopic: Plate bed compact fibrous tissue with occasional macrophages in clefts and about blood vessels.

Dog 56.—*Operation:* August 19, 1940. Four screws inserted in parieto-occipital region. Three screws inserted into femur. Healed *per primam.* Sacrificed September 25, 1941.

Macroscopic: Operative site insignificant. All 7 screws firmly embedded. Metal shiny. No fluid.

Microscopic: Femur: Screw tract lined by thin fibrous tissue. Fine trabeculae of new bone aligned beneath part of tract lining. In intervals where bone is deficient fibrous lining lies on fatty marrow. Skull: Screw holes lining shows no inflammation or necrosis.

Dog 57.—*Operation:* August 23, 1940. Four screws inserted into frontoparietal region. Four screws inserted into femur. Healed *per primam.* Sacrificed September 24, 1941.

Macroscopic: Operative scars grossly negative. Eight screws firmly embedded. Metal smooth and shiny.

Microscopic: Screw tracts in both bones show no inflammation or necrosis. Thin fibrous layer

FIG. 2.—(Dog 57)—"Wrought" Ticonium: Screw hole in skull. Eleven months after operation. (H. and E., ×170)

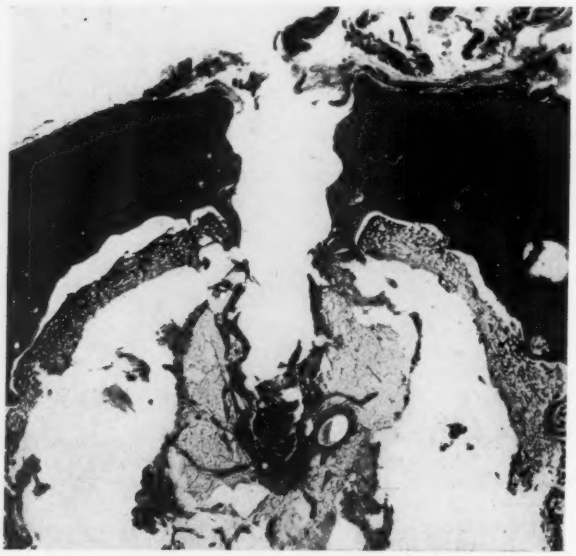

FIG. 3.—(Dog 56)—"Wrought" Ticonium: Screw hole in femur. Eleven months after operation. (H. and E., ×14)

over head of screws in skull and a spur of new bone is present in thickened periosteum of same region.

Dog 58.—*Operation:* August 22, 1940. Four screws inserted into frontoparietal region. Three screws inserted into femur. Healed *per primam.* Sacrificed September, 1941.

Macroscopic: Operation scars not remarkable. Screws firmly embedded. Metal shiny.

Microscopic: Screw tracts lined by thin fibrous layer with rare pigmented macrophages nearby.

Dog 64.—*Operation:* August 28, 1940. Plate secured with 4 screws. Post. lat. screw not inserted in full length. Flap healed *per primam.* Sacrificed September 23, 1941.

Macroscopic: Operative scar not unusual. Plate firmly enclosed in bursa-like sac. Screws tight. Metal smooth and shiny. Bony defect filled by fibrous layer. Dura not adherent.

FIG. 4.—(Dog 43)—"Wrought" Ticonium: Brain showing the tissue immediately adjacent to the embedded piece of metal. Eleven months after operation. (PTAH, ×170)

Microscopic: Plate bed consists of usual 3 layers of thin partly hyalinized collagen with rare pigment bearing macrophages in clefts about blood vessels. No inflammation or necrosis.

Dog 67.—*Operation:* August 20, 1940. Four screws inserted into frontoparietal region. Three screws inserted into femur. Healed *per primam.* Sacrificed September 24, 1941.

Macroscopic: Operative scars not unusual. Seven screws firmly embedded. Metal smooth and shiny.

Microscopic: Screw tracts in both sites lined by thin fibrous tissue layer. No inflammation. No necrosis.

Dog 68.—*Operation:* August 20, 1940. Dura vascular. Dura opened. Plate secured with 3 screws. Flap healed *per primam.* Sacrificed September 25, 1941.

Macroscopic: Operative site not unusual. Plate firmly embedded into usual bursa-like sac. Screws tight. Dura merges with fibrous tissue filling bone defect. Underlying cortex negative.

Microscopic: Plate bed smooth thin compact fibrous layer with slight new bone formation in bony defect. Rare pigmented macrophage.

Dog. 70.—*Operation:* August 31, 1940. Four screws inserted into frontoparietal region. Three screws inserted into femur. Flap healed *per primam.* Sacrificed September, 1941.

Macroscopic: Operative scars not significant. All screws firmly imbedded; shiny.

Microscopic: Screw tracts in both bones have thin fibrous linings resting on fatty marrow or spicules of newly formed bone. Some periosteal new bone also formed around screw point in skull where it has passed through internal table.

COMMENT: All screws and plates in this series were securely fixed and showed no corrosion.

In the presence of a wrought Ticonium plate, skull defects underwent uncomplicated repair. Two layers of parallel hyalinized fibers were laid down continuous with the pericranium and dura, respectively, the pericranial layer being somewhat thicker. Between these two layers was a third, the new fibers of which were deposited parallel to the surface but at right angles to those of the other two. The larger blood vessels coursed in this middle layer. A limited amount of periosteal new bone was formed which failed to fill the gap. The bone margins underwent remodelling so that they were smooth and tapered in cross-section. Small tufts of muscle filled the plate perforations. Screw holes were lined by a thin layer of fibrous tissue with fibrils again parallel to the metal surface. This lining lay on a base composed of either new bone spicules or fatty marrow (Figs. 2 and 3).

The tissue about metal implants in the brain showed only a thin layer of fibrous tissue beneath which were a few macrophages. The brain substance proper showed a narrow zone of gliosis. The vessels appeared negative (Fig. 4).

A single exception to the otherwise uniform results was found in Dog 46. Necrosis and polymorphonuclear cell exudate characterized the tissue response. This was unique in this group, which included 21 dogs and 115 pieces of metal. The reaction in this case could be differentiated, histologically, from the toxic response seen in Group III. We could not, however, differentiate between Dog 46 and one infected case in Group III. The clinical history and the pathologic findings, as noted in the protocol, suggested infection.

In many cases, a dark brownish or black pigment containing iron was present in macrophages. These were found in clefts between the fibrous tissue of the skull defects, just beneath the fibrous lining of screw holes and in the muscle about the screw heads. The macrophages were most numerous about blood vessels but could not be found immediately adjacent to the metal. The amount of pigment was small, never constituting granulomata. It appeared to be identical with the pigment seen about Vitallium plates and screws.

It may be concluded, therefore, that wrought Ticonium is essentially inert, as is Vitallium.

GROUP II: VITALLIUM

This alloy was examined in seven dogs. The tissues were exposed in all to a total of 34 pieces of metals; three plates and 31 screws. The animals were observed from 24 days to 15 months. Five dogs were sacrificed, and two died on account of secondary illness.

CASE REPORTS

Dog 6.—*Operation:* December 1, 1939. Plate secured with 3 screws. Secondary subcutaneous infection in area about medial flap margin. Healed 26th p.o. day. Sacrificed September 3, 1940.

Macroscopic: Skin scar sound. Muscle tufts have grown through perforations in plate. Screws tight. Plate firm. Metal smooth, and shiny. Dura thickened but not adherent to cortex.

Microscopic: Plate base consists of hyalinized fibrous tissue merging with bone. At the margin

of the two tissues are a few spicules of new bone arising from periosteum. Very rare macrophage in fibrous tissue clefts contains iron pigment.

Dog. 13.—*Operation:* December 29, 1939. Plate secured with 3 screws. Healed *per primam.* Sacrificed September 3, 1940.

Macroscopic: Skin scar negative. Plate and screws tight. Muscle has grown through plate perforations. Metal smooth and shiny. Dura thickened beneath the defect but not adherent to cortex.

Microscopic: Defect filled with bridge of hyalinized fibrous tissue merging with bone. In clefts of fibrous tissue iron-containing pigment in macrophages. Muscle attached to plate bed appears negative. Some foreign body response to bone wax.

Dog 16.—*Operation:* February 15, 1940. Solid plate secured with 3 screws. Posterior screw not tight. Flap healed *per primam.* Sacrificed September 3, 1940.

Macroscopic: Skin scar sound. Screws firm. Plate shiny and well fixed. Underlying dura thickened but not adherent to cortex.

Microscopic: Defect bridged by laminated, partly hyalinized fibrous tissue. Between a few fibers, especially near vessels, are occasionally pigment bearing macrophages. Slight new bone formation.

Dog. 30.—*Operation:* June 29, 1940. Six screws inserted into frontoparietal region. Due to injury, flap broke open. Secondary subcutaneous purulent infection. Healed 20th p.o. day. Sacrificed September 23, 1941.

Macroscopic: Scar and musculature negative. All 6 screws firmly embedded, smooth and shiny.

Microscopic: Screw tract lined by thin fibrous layer, with fibers parallel to screw surface. This rests on spicules of new bone or on fatty marrow. Considerable periosteal new bone at dural end of tract, where screw has evidently penetrated inner table pushing periosteum before it. Few pigment-laden macrophages in looser connective tissue just beneath fibrous lining. Same type of macrophages in connective tissue about head of screw. Here they seem chiefly perivascular.

Dog 31.—*Operation:* June 30, 1940. Six screws inserted into frontoparietal region. All screw holes somewhat too large. Flap healed *per primam.* 26th p.o. day, drainage from eyes and nasal mucous membranes. Slow and incomplete recovery. Two months later flaccid paralysis of both hind legs. Epileptic seizures. "Distemper." Died December 19, 1940.

Macroscopic: Operative scar negative. Six screws firm.

Microscopic: Screw tract lined with thin laminated fibrous tissue. Occasional pigment-laden macrophage in looser tissue immediately adjacent.

Dog 32.—*Operation:* July 6, 1940. Six screws inserted into frontoparietal region. Flap healed *per primam.* 5th p.o. day, drainage from eyes and nasal mucous membranes, fever, dyspnea. "Distemper." Died August 1, 1940.

Macroscopic: Operative scar insignificant. Muscle grossly negative. Six screws firm and shiny. All 6 screws have passed through the inner table. Appear as firm smooth nodules on dural sides. Not adherent to cortex. Right atrium and ventricle of heart dilated. Lungs consolidated.

Microscopic: Screw tract lined with thin fibrous tissue layer. Considerable periosteal new bone formed at dural end of tract. Rare pigment bearing macrophage.

Dog. 34.—*Operation:* July 10, 1940. Four screws inserted into frontoparietal region. Flap healed *per primam.* Sacrificed September 23, 1940.

Macroscopic: Muscle and skin of operative site negative. Four screws firm. Metal smooth and shiny. Dura smooth.

Microscopic: Slight new bone formation between dura and inner table near point of screw. No reaction in tract.

COMMENT.—The original purpose of using Vitallium in these experiments was chiefly to supply a control series. These results are reported merely to confirm the histologic studies of Venable, Stuck, and Beech.[2, 3] They are of particular interest only in that a longer observation and survival period is covered than yet recorded.

In brief, no evidence of toxicity was found. The screws were tight, the plates firmly fixed, and the metal shiny. Cranial defects healed normally, and the tissues on which the plate rested showed no necrosis nor inflammation. The screw holes were lined by a thin layer of fibrous tissue overlying newly formed trabeculae of bone or fatty marrow (Fig. 5).

In every case an occasional pigment-bearing macrophage in the vicinity of the metal was observed. These cells were located in spaces between collagen fibers in the skull defects and about blood vessels. In screw hole tracts they were usually seen beneath the thin fibrous inner lining, and here, too,

were often clustered about vessels. They were not noted immediately adjacent to the metal. This pigment contained iron, and it is presumed to be the same as that reported by Wise[9] about a Vitallium Smith-Petersen nail. It is worthy of note that this pigment occurred in very small amounts, that it did not give rise to granulomata, and in our opinion, does not constitute a sign of toxicity. As was noted above, this pigment was also found in

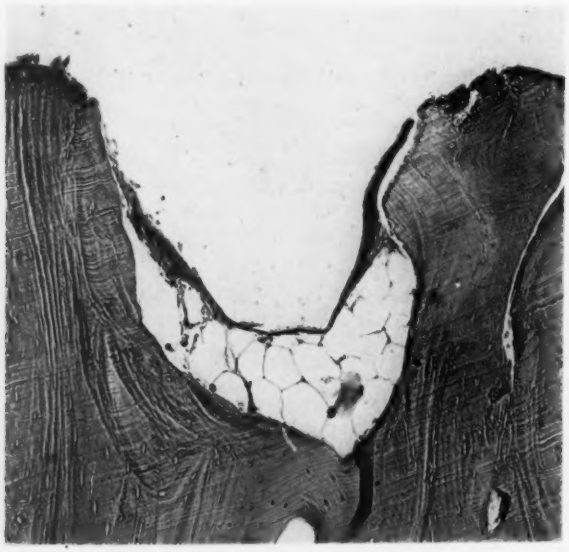

FIG. 5.—(Dog 30)—Vitallium: Screw hole in skull. Fourteen months after operation. (H. and E., ×170)

animals in which Ticonium "wrought" was buried. Since both Vitallium and wrought Ticonium are nonferrous alloys, the iron cannot have been derived from these metals. Our data do not permit us to assign its source. We are inclined to agree with Wise that it is of hematogenous origin.

GROUP III: TICONIUM "CAST" WITH BERYLLIUM

This alloy was tested in 14 animals. The tissues were exposed to a total of 55 pieces of metals: Nine plates, 35 screws in the skull bone, 10 screws in the femur, and one piece of metal which was embedded into the cerebral cortex. The dogs were observed from 50 days to 22 months. Eight animals were observed for a period longer than 10 months. Eleven dogs were sacrificed, three died on account of secondary illness.

COMMENT.—The findings in this group may be summarized without giving the individual case reports of the 14 animals observed.

In dogs which were killed before six months, plates were firmly fixed, screws were tight and the metal was bright and shiny. In dogs which survived longer, however, screws were usually loose and the tissues about the plates were discolored. Occasionally, fluid was found about the metal.

Histologically, the screw holes and plate beds were lined by chronically inflamed granulation tissue containing many macrophages. In the vicinity of the screw holes there was extensive fibrosis of the marrow and some

new bone formation. In general, the amount of lymphocytic and macrophagic exudate was greater in long term experiments. In one dog which survived three months the plate bed consisted of dense fibrous tissue in which were a few focal collections of lymphocytes and macrophages especially numerous about the plate edge. In another dog which survived 22 months, the lining of the space filled by the plate consisted of macrophages with their long axis perpendicular to the plate surface so that these formed a palisade-like layer. The tissue beneath them was highly vascularized and great numbers of swollen macrophages and lymphocytes were enmeshed in a loose fibrillar network. There was a thick layer of fibrous tissue, partly hyalinized beneath the granulations. In the interstices of this were foci of lymphocytic infiltration. Similar changes were seen in the brain where a thick fibrous capsule was surrounded by degenerated brain substance markedly infiltrated with lymphocytes. Perivascular lyphocytic collars were present at some distance from the metal. In spite of the extensive exudative reaction, fibroplasia and osteoplasia were not significantly impaired. In the skull periosteal new bone formed near the bony margin of the defects. In femurs the usual fine trabeculae of the marrow were replaced, in the vicinity of the screws, by dense bone having well developed haversian systems.

These observations are at variance with the results obtained in fibroblast cultures. Two explanations suggest themselves. The first is that the time during which a metal may be studied in any one generation of fibroblasts *in vitro*, is insufficient. Against this explanation is the unimpaired fibroplasia in the long term *in vivo* experiments. The second is that a factor of *selective cytotoxicity* complicates the picture. By this it is meant that some metals may not be toxic to fibroblasts although they may be toxic to other types of cells.

Whatever the explanation, Ménégaux's[8] belief that fibroblast cultures are a valid medium for testing all metals may be questioned.

OBSERVATIONS ON ELECTROLYSIS

Venable, Stuck, and coworkers[2, 3, 4] maintain that electrolysis is the controlling factor in osteosynthesis with metals, and that "metals which are nonelectrolytic in body fluids cause no pathologic reaction in the tissue." This has been a point of controversy. Murray, Martin[10] (Fracture Committee of the American College of Surgeons, New York, 1938), and Key[11] do not feel that electrolysis has an essential bearing on the problem. Bothe, Beaton, and Davenport [12, 13] concluded that "electrolysis is an accompaniment of unfavorable bone reaction rather than the direct cause of it."

Using Venable's[3] method, we have checked the electrolytic activity of the two Ticonium alloys and compared them with that of Vitallium and Vanadium. Silver and copper were used as anodes. Ringer's solution served as electrolyte. A micro-ammeter with an internal resistance of 50 ohms was used. Full-scale deflection amounted to 120 micro-amps. The results are given in Table I.

It is of interest to note that the final reading for Ticonium "wrought" and Vitallium approximates zero, while Ticonium "cast" shows only a minimum current. Ticonium "cast" did not prove to be toxic to fibroblast cultures but did give a local toxic response in dogs which was out of all proportion to the minimal galvanic current produced.

It is common knowledge that pure copper is toxic by chemical action even though it cannot be accused of being electrolytic. This may be taken to indicate that electrolysis may be an accompanying factor of unfavorable bone reaction but it cannot be its only cause. Key has presented good evidence as to its lack of importance clinically.

TABLE I

ELECTROLYTIC DETERMINATIONS

Cells in Ringer's Solution	Initial Deflection in Micro-amperes	Immediate Drop in Micro-amperes to	Deflection after 3 Minutes in Micro-amperes	Deflection after 90 Minutes in Micro-amperes
Ticonium "wrought"-copper	24	3	0	0
Ticonium "wrought"-silver	24	3	0	0
Vitallium-copper	35	2½	0	0
Vitallium-silver	35	2½	0	0
Ticonium "cast"-copper	100	24	13	0
Ticonium "cast"-silver	100	24	13	0
Vanadium-copper	"off scale"	—	—	"off scale"
Vanadium-silver	"off scale"	—	—	"off scale"

The data recorded in this table were compiled by Mr. E. Griffith, Chief-Metallurgist, Research Laboratories, "Ticonium" Co., Albany, N. Y.

Discussion.—Our observations, as expressed in the present communication, establish the inertness in tissues of wrought Ticonium and Vitallium.

Contrary to our previous conception, the cast alloy Ticonium (containing beryllium) proved to be toxic *in vivo*.* It did not, however, interfere with fibroplasia or osteoplasia in any of the animals observed. Therefore, it seems unlikely that the short duration of the tissue culture experiment is the explanation for the lack of "fibroblast cytotoxicity." The evidence is in favor of a *selective cytotoxicity* of the toxic agent, which presumably in this case is beryllium.

Our experiments to date lead us to believe that fibroblast cultures and electrolytic measurements are inferior to histologic studies as tests for the toxicity of metals.

In the alloy Ticonium "wrought" (*without* beryllium), it is believed that a material has been found which conforms to the requirements set forth for the use of metals in the repair of cranial defects. Accordingly, we have commenced to use this alloy as cranioplastic material in patients and will report our observations at a later date.

* Ticonium is made in several forms, "wrought" Ticonium for surgical purposes, and a cast form with beryllium for dental restorations. As employed in the dental alloy, beryllium, we are informed, improves the casting qualities of Ticonium and assists the production of appliances of extreme dimensional accuracy. It has been used successfully in dentistry for some years. Surgical appliances are not made of cast Ticonium with beryllium. While this alloy is toxic *in vivo*, no inference can be drawn from our experience and information on its behavior in such external appliances as dental restorations.

SUMMARY

1. The alloys Ticonium "wrought," Vitallium, and Ticonium "cast," have been studied *in vivo* with special reference to their usage as cranioplastic material.

2. Ticonium "wrought" and Vitallium have been found inert. Ticonium "cast" (with beryllium) has been found cytotoxic. It is suggested that this toxicity is "selective."

3. Fibroblast cultures and electrolytic measurements have been found inferior to histologic studies as tests for the toxicity of metals.

4. The importance of carrying out *in vivo* studies for periods longer than six months is indicated by the late toxic reaction in the Ticonium "cast" material.

5. The wrought alloy Ticonium appears worthy of trial as a cranioplastic material in man.

The authors wish to express their appreciation to Drs. B. Williams and T. Engster for their assistance, and to Mr. E. Touceda and Mr. E. Griffith for their advice in the metallurgic phases of the study.

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THE USE OF A LAMP TO WARM MOIST COMPRESSES*

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HOW TO KEEP COMPRESSES HOT is a perennial question on any surgical service. Its answer interests the nurse, administrator, and surgeon, for practical technic, hospital costs and effective therapy are involved. The control of temperature probably influences the rate of healing of any wound but is especially important in lesions of an extremity having an impaired circulation, for here uncontrolled heat may cause harm.

Compresses are usually kept at a higher temperature than the surrounding air, so some external source of heat must be provided to counteract the normal heat loss. The five methods of supplying heat that have been used are: (1) The repeated application of a hot solution or frequent changes of hot dressings. (2) A poultice. (3) Hot water bottles. (4) An electric heating pad. (5) Radiant heat from a lamp. The repeated application of a hot solution or hot dressings is inefficient because of rapid cooling, after which the compress becomes cold and clammy. The preparation of a poultice is rapidly becoming a lost art and, though those prepared by some older nurses appeared to do good, yet one hesitates to order them at present for fear of the result. Hot water bottles are safe and, if covered with a rubber sheet and blankets to prevent heat loss, are effective. They have the disadvantages of causing pain in an infected wound from the weight of the bulky dressing and of requiring excessive nursing time to keep them warm. Electric pads would appear to be satisfactory, but the usual ones cannot be relied upon to maintain a constant temperature, so are a source of trouble and worry. A satisfactory heating element can be manufactured at a moderate cost, but a good rheostat cannot be made for the price obtained for most electric pads. The use of an accurate rheostat has been tried but it increased the cost beyond the salability of the product and was discontinued. Cooley[2] has described an excellent electric heating unit designed for compresses. It has an accurate, controllable rheostat, but this very fact makes it expensive for the average institution to purchase the number required for an active surgical service.

Lights or baking lamps have long been used in treatment, and Dr. Stafford Warren† and I thought radiant heat from this source would be the cheapest, safest, and most effective means of heating compresses. The only question to be settled was how it could best be applied. A bed cradle, with lights controlled by a rheostat, will maintain a fairly constant temperature but it is awkward and bulky for routine use on compresses. The portable baker

* This study received assistance from the Siever's Fund.

† Doctor Warren first interested me in the use of radiant heat when I had an infected hand, and could judge, subjectively, the effect of various compresses. I am indebted to him for advice during these observations.

used in physiotherapy gives a concentrated heat to a limited area. It has two drawbacks for unrestricted use: First, high initial cost; and, second, danger from burns. The "therapy" or "baking" lamp is easy to regulate, cheap, and compact, so appeared to be the best means of supplying the radiant heat to compresses.

The construction of such lamps is fairly uniform, most of them having an aluminum reflector with either a tungsten bulb or a silicon-carbon heating element. They vary in design, and in their external frame, for there are "floor" models, "table" models and "hand" or bracket lamps. None of these commercial types were suited for our purpose. The "floor" model with a round base was unstable and would tip over, while that with a tripod base was cumbersome to have at the bedside since the legs stuck out and the attendants tripped over them. The "table" model has a very small base which made it so unstable that it was dangerous. If one of these hot lamps fell onto a patient it would cause a severe burn; so that a rigid attachment was considered obligatory, it being better than insurance.

Hand lamps with a spun aluminum reflector and tungsten bulbs were purchased for $2.48 each, and stripped of their handles. A ¼″ rod, 18″ long, was welded to the ball and socket joint of the bracket and fastened by a laboratory clamp holder to a ⅜-inch upright rod, 36 inches long. This upright rod was fastened to a specially machined bracket which clamped onto the frame of the bedspring (Fig. 1). This bracket allowed adjustment of the lamp to any point from the head to the foot of the bed, while the laboratory clamp connecting the vertical and horizontal rods gave adjustment in rotation up and down, or across the bed; yet, once it was tightened in place the whole apparatus was rigid and secure, being free from the danger of falling on the patient. This arrangement was cheap (total cost $7.50 per unit), compact, and appeared satisfactory; but when it was put to use an unexpected difficulty arose from variation in bedspring construction. It was found that in this hospital alone there were five different kinds of bed springs! These were of two general types, one with the angle-iron of the frame turned up and the other turned down, so, with minor adjustments two types of the bracket could be made to serve (Fig. 2).

In an effort to construct an attachment suitable for all beds the idea of clamping the lamp to the spring was discarded in favor of attaching it to a rod which was clamped to the head and foot of the bed (Fig. 3). The lamp assembly was mounted on a sleeve which slid on a piece of pipe-conduit attached to the ends of the bed. For ease of attachment a hook was put on the head-end to hold it in place, while the screw was tightened at the foot. This apparatus cost about $10.00, when made in our shop. It was a little unwieldy to carry about the hospital but, other than this, has been satisfactory.

NURSING METHOD

The routine use of these lights for compresses is as follows:

(a) The bed is protected with a rubber sheet.

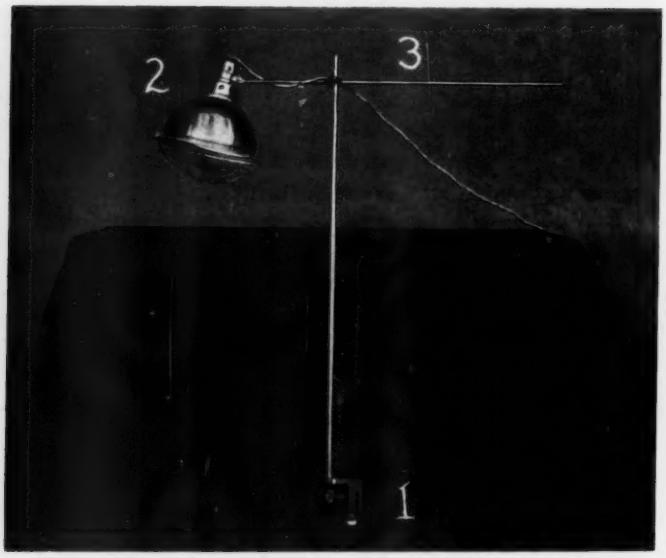


FIG. 1.—The compress light which attaches to the bed. (1) Shows one type of the machined clamp which attaches to the angle-iron of the bed spring to hold the vertical 36″ rod. (2) Is the lamp which has the ball and socket joint of its original bracket welded to a horizontal rod 18″ long. (3) Shows the attachment of the horizontal rod to the vertical rod by a laboratory clamp-holder, which gives vertical, horizontal, and rotary motion.

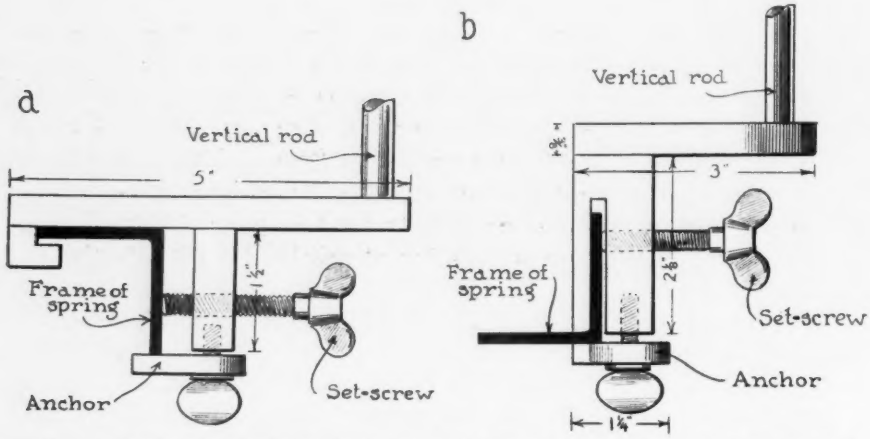
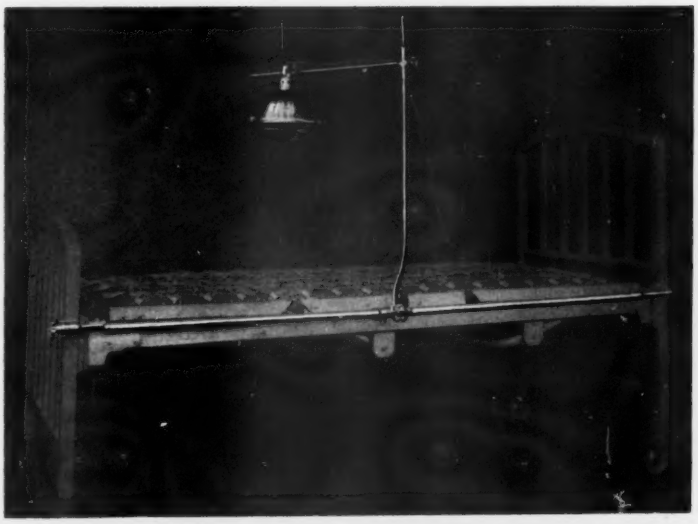


FIG. 2.—Construction details of the two types of clamps to fasten the vertical rod to the bed spring. (a) Shows the clamp for use when the angle-iron of the frame turns down. Adjustment is provided for 1½″ and 1¾″ angle-iron. The button or "anchor" at the bottom was found to add to the stability. (b) Illustrates the clamp designed for a frame with the angle-iron turned up. The slot slips over the frame and is held with a set-screw. Here, also, adjustment is provided for variation in the size and thickness of the frame.



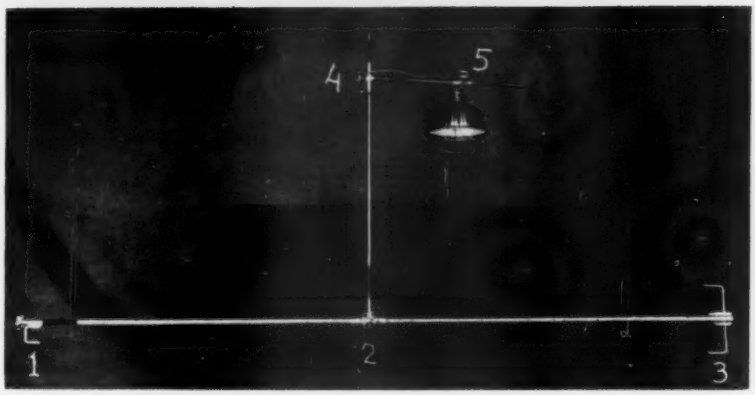


FIG. 3.—Attachment of the lamp to a pipe which is clamped to the head and foot of the bed. In the lower illustration (1) is a clamp tightened by a screw that threads on a nut inside the conduit. This screw is 12″ long to allow adjustment for beds that vary in length from 6′6″ to 7′4″. (2) Is a sleeve that slides along the pipe to the head or foot of the bed. It carries a vertical rod 36″ long, which has a slight outward bend at its base to clear the mattress. (3) Is a hook to catch on the cross-bar of the head of the bed to hold this clamp while the other end is tightened. It was originally made with two hooks, to be interchangeable on the two sides of the bed. (4) Is a sleeve that fastens on the vertical rod and carries the horizontal rod. This raises, lowers, or rotates the lamp. (5) Is a sleeve which allows movement in a horizontal plane or permits tilting the lamp

(b) The wound is dressed with two layers of moist gauze covered with oiled silk.
(c) This dressing is covered or wrapped with a sterile towel.
(d) A bath thermometer is placed near the wound.
(e) The light is centered on the wound and raised or lowered until the thermometer reads 110° F.
(f) Unprotected skin near the lamp may be covered with a towel to prevent discomfort.
(g) Drafts near the lamp are avoided.
(h) Compresses are moistened every three hours.

The temperature beside the wound may be from 5° to 10° F. lower than that of the bath thermometer outside the compress.

RESULTS

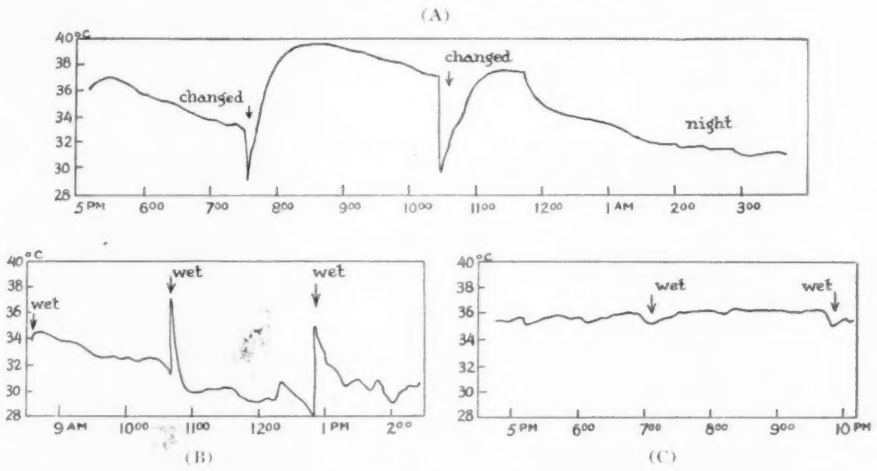
After using these lamps for a year, it has been observed that when moist compresses are used the nurses prefer the lamp because it takes less work, the patients like it because it is comfortable, and the surgeon orders it because it is effective. However, these opinions are partially subjective, so it was desired to obtain quantitative information on the temperature control and nursing time with the various types of compresses.

The temperature was obtained by means of an electrode separated from the wound by one layer of gauze. This electrode was attached to a continuous temperature recording device loaned to me by Mr. Bishop. Typical graphs are shown in Graph 1, where it will be seen that only with the light is a continuous elevation of temperature maintained. This has also been observed by Cochran.[1] The "massive compress" consists of moist gauze, covered by oiled silk, wrapped with a Turkish towel, which has several hot water bottles outside it. These are covered with a rubber sheet, and the whole is wrapped in a blanket which is pinned tightly in place from the tip to the base of the limb. With all this, the temperature is found to fall after about an hour (Graph 1A). Dressings which are moistened periodically with a warm solution (Graph 1B) are found to cool in 10 or 15 minutes to room temperature. Compresses warmed with a lamp fluctuated in temperature only if the patient's bed was in a draft.

Computation of nursing time for the care of different compresses gave interesting results. The head nurses kept an accurate record of the time consumed with the different procedures. The "massive compresses" required an average of four hours a day, per patient, or 28 hours a week. The moist compresses, kept warm by a lamp, required an average of 40 minutes a day or 4.66 hours a week, or a saving of 23.33 hours a week of nursing time per patient. If nursing time is considered to cost an average of 60 cents per hour, then the "massive compress" costs the hospital $16.80 per patient per week for nursing, while the compress and light cost $2.80, or a saving of $14.00 per patient week. The added cost of the electric current used for the lamp about equalled the cost of the added dressings used in the "massive compress." The amount saved in nursing time in one week on

one patient was more than the initial cost of the lamp; so the administration of the hospital was very willing to buy all the lamps that were needed.

This study brought up a question that was not satisfactorily answered—"What is the optimum temperature for the healing of a wound?" We know that the normal vasodilatation level is between 31° and 33° C.[3] We also



GRAPH 1.—Continuous temperature readings on moist compresses. (A) "Massive compresses"—Nursing time, four hours a day. The temperature is maintained for about an hour and then begins to fall. They are changed every three hours. (B) "Moist" dressings without external source of heat. Moistened every three hours. Nursing time, 40 minutes a day. The heat is retained for only 10 or 15 minutes. (C) "Moist" dressings, with light as source of heat. Moistened every three hours. Nursing time, 40 minutes a day. A constant, effective temperature is maintained.

know that in peripheral vascular disease this level is as low as 23° or 24° C., and that overheating such an extremity accumulates waste products from local tissue metabolism which damages the tissue. This demands exact control of the external temperature with an impaired circulation. But with a normal blood supply it is possible that the rate of healing of wounds can be accelerated by temperatures in excess of the normal vasodilatation level. This should be determined, for it is pertinent.

SUMMARY

Warm, moist compresses are a satisfactory means of treating open, infected or granulating wounds. For this purpose, a source of external heat must be provided. A baking lamp was found to maintain a constant temperature, to eliminate the bulk and weight of a large dressing and to save nursing time. One year's experience has shown it to be safe and effective.

Clamps to attach these lamps to the bed have been designd and are described.

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THE ANATOMIC APPROACH TO PULMONARY RESECTION

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INDIVIDUAL LIGATION of the pulmonary vessels and careful suture of the bronchus in the performance of complete unilateral pulmonary resection has received preference over other measures for several years. This method was popularized by Rienhoff,[1] Crafoord,[2] Mason[3] and others and the results have been so satisfactory that few will resort to tourniquet control of the

FIG. 1. A

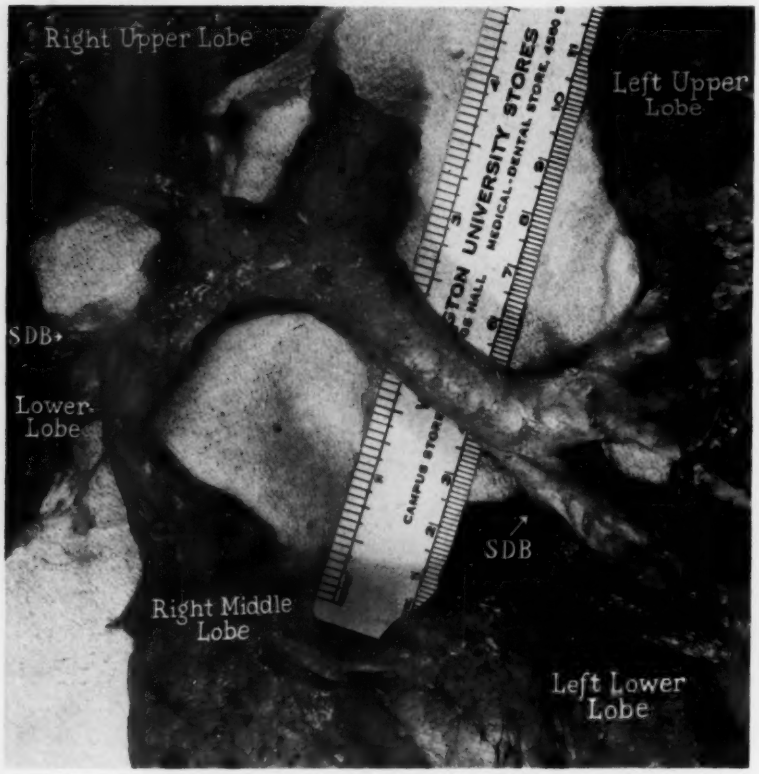

FIG. 1. A.—Dissected specimen showing bronchial divisions of greatest surgical importance. Note branch bronchus to superior divisions of the lower lobes (SDB).

hilum or to mass ligation except in those instances in which individual dissection of the pulmonary veins, the pulmonary artery and the bronchus are impossible or unwise because of existing circumstances. More recently, efforts have been made to apply the principle of separate ligation and suture to partial pulmonary resection by ourselves[4, 5] and by Churchill.[6, 7] Here again, the preliminary results have been so promising that the technic will, in our opinion, replace all other methods save in those cases in which it cannot be employed because of technical or other reasons.

Combined clinical experiences and investigatory observations have yielded certain facts of anatomic importance which we consider worth making a part of the record. The comprehensive picture of the surgical anatomy of pulmonary resection may be assembled upon our conception of the tracheobronchial tree. In addition, the management of the bronchial stump after pulmonary resection is so important that the anatomic characteristics of the bronchial tree are secondary to no other considerations.

FIG. 1. B

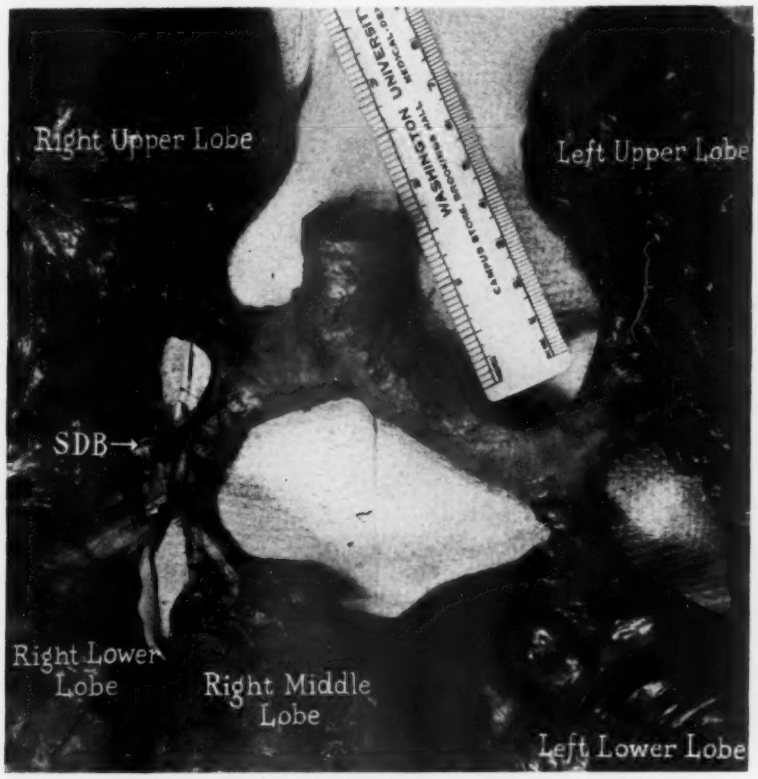

FIG. 1. B.—Another specimen showing similar pattern of the bronchial tree. This is the configuration usually encountered.

Figure 1, A. and B., are photographs of dissections of lung specimens made to demonstrate the surgical anatomy of the bronchial tree. An understanding of these structures is important, whether it is to be applied to total unilateral or to partial pulmonary resection. The similarity of the specimens is striking and characteristic. It can be seen that the left main bronchus is so long that it offers more than adequate proportions for amputation and complete closure of the stump except in those instances in which it is necessary to divide the bronchus at the trachea. On the right side, the main bronchus is shorter, due to the point of origin of the upper lobe bronchus. Indeed, it is true that the upper lobe bronchus sometimes arises at a much higher level than shown in the illustration, and, occasionally, even from the wall

of the trachea. In general, however, the main bronchi are entirely adequate for purposes of surgical division and repair.

A study of the secondary or lobar bronchi is next in order so that these observations may be applied to lobectomy performed by the principle of individual handling of the hilar structures. As far as the bronchi are concerned, it is obvious that both upper lobes and the right middle lobe afford structures of sufficient length to allow amputation with subsequent closure of the stump. The lower lobes, however, present a somewhat different problem, especially on the right side. Fig. 1, A. and B., shows

FIG. 2. A

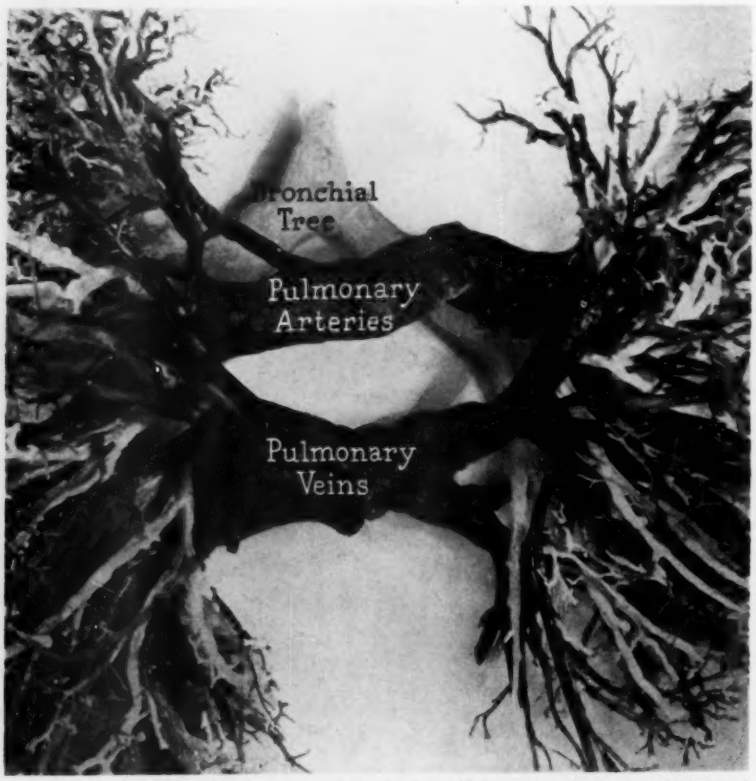

FIG. 2. A.—Injection-mass cast of tracheobronchial tree, pulmonary arteries and pulmonary veins (anteroposterior view).

the relationships of the lower lobe bronchi and it is important to study the secondary bronchi which lead to the apices or superior divisions of the lower lobes. These bronchi are highly important for they represent the primary subdivisions of the lower lobe bronchi and also because it is sometimes possible, as pointed out by Churchill and Belsey,[6] to resect either the superior division of the lower lobe alone or to remove the remainder of the lower lobe while leaving the superior division of the lower lobe intact. In performing complete lower lobe resection, the main bronchus to the lower lobe must be transected above the point of origin of this branch bronchus to the apex of the lobe. This is of particular importance on the right side because the

branch bronchus usually arises at about the level of the middle lobe bronchus, the former springing from the posterior and the latter from the anterior aspect of the main bronchial axis. If the right lower lobe is to be resected, the middle lobe bronchus must be preserved and must not be obstructed as a result of the removal of the lower lobe. It may, therefore, be necessary to divide the bronchus to the apex of the lower lobe separately, before the main trunk of the lower lobe bronchus at a point distal to the apical branch bronchus is exposed.

The surgical importance of the apices of the lower lobes cannot be overestimated. They have been aptly termed "a lobe within a lobe" because of the anatomic independence of the structures. Indeed, during an extensive study of many heart-lung specimens made at autopsy, the single outstanding anomaly has been one instance of three discrete lobes on the left.

FIG. 2. B

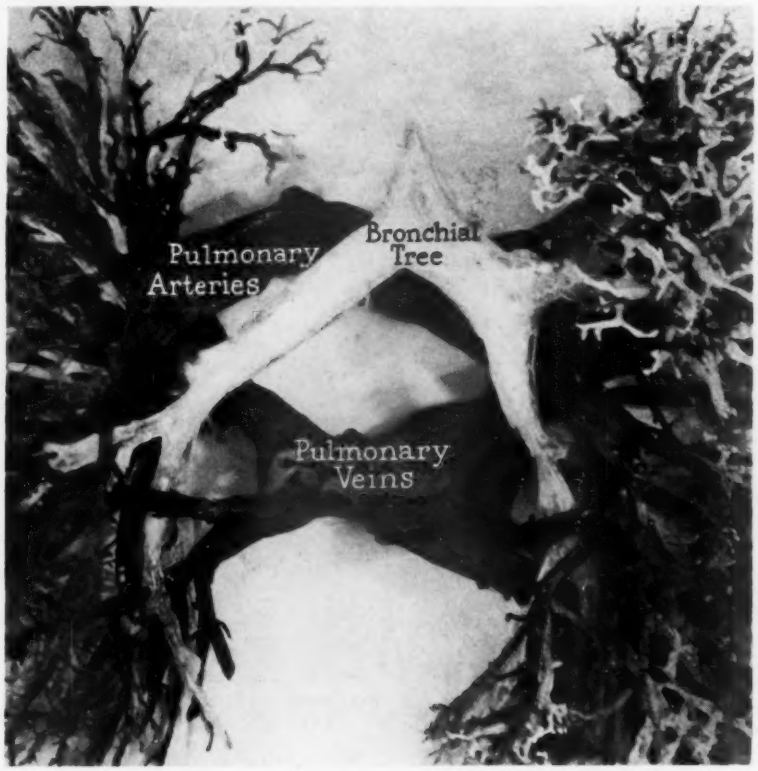

FIG. 2. B.—Same from postero-anterior view.

The preparation of several injection-mass specimens has aided in the establishment of the relationships of the pulmonary arteries and veins to each other and to the bronchial tree. The lumens of these structures were filled with a synthetic resin of varied colors, the tissues then being subjected to digestion by acids. Figure 2, A. and B. show the result of one such attempt. The principal information to be gained is orientation of the components

to each other. It can be seen that the pulmonary veins are anterior and inferior to the pulmonary arteries, which in turn are anterior to the right and left main bronchi. Study of the actual preparations yields a third dimension concept of the orientation of the important secondary and tertiary subdivisions of each major structure, but this cannot be demonstrated successfully in photographic illustrations. However, the pulmonary veins are well shown in Figure 2 and the relationships of the superior and the inferior pulmonary veins, the branches from the right middle lobe, the branches from the lingula, and the apical branches from the lower lobes to the inferior pulmonary veins are all clearly depicted.

FIG. 3. A

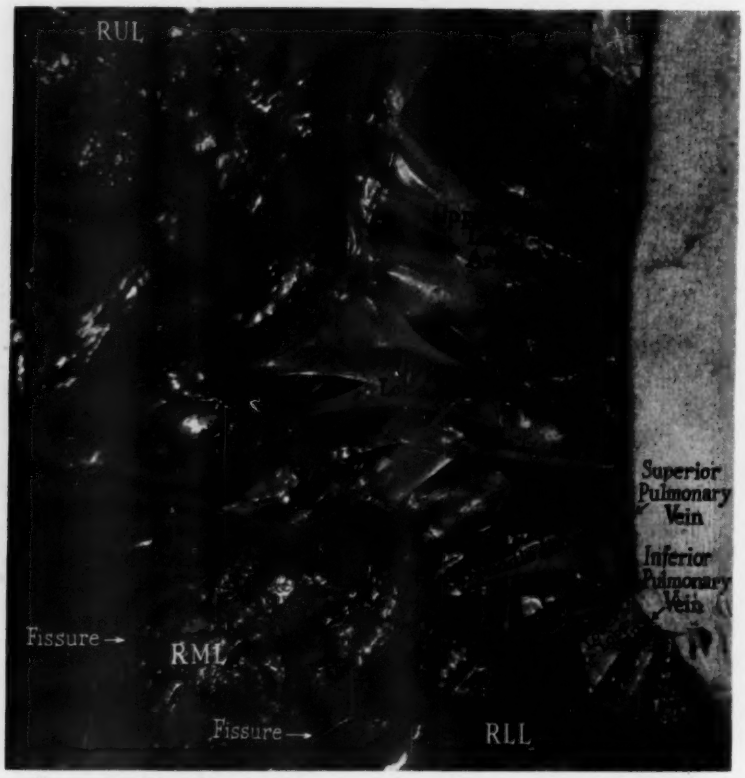

FIG. 3. A.—Dissection of right lung root, anterior aspect. The relationship of the pulmonary veins, the right pulmonary artery and the right main bronchus are emphasized.

Figure 3, A. illustrates the structures of the right pulmonary hilum as viewed from the anterior aspect. The superior pulmonary vein with its tributary from the middle lobe is clearly demonstrated and the inferior pulmonary vein from the lower lobe can be seen without difficulty. It is our belief that pulmonary resection may be performed more safely if the veins are disposed of first because the avenue of entry for air or other embolic matter is thus closed. The veins lie on the anterior surface of the

hilum just beneath the pleural reflection which covers the lung root. The superior pulmonary vein is easily located, but it is sometimes more difficult to isolate the inferior pulmonary vein. The inferior vein, either right or left, lies upon the anterior surface of the respective inferior pulmonary ligament close to the hilar end of the ligament. Pursuit of the inferior pulmonary ligament, therefore, has been found to offer an excellent approach to the inferior pulmonary vein. Figure 3, A. also reveals the relationship of the arterial branch to the right upper lobe, to the veins, and to the right main bronchus.

FIG. 3. B

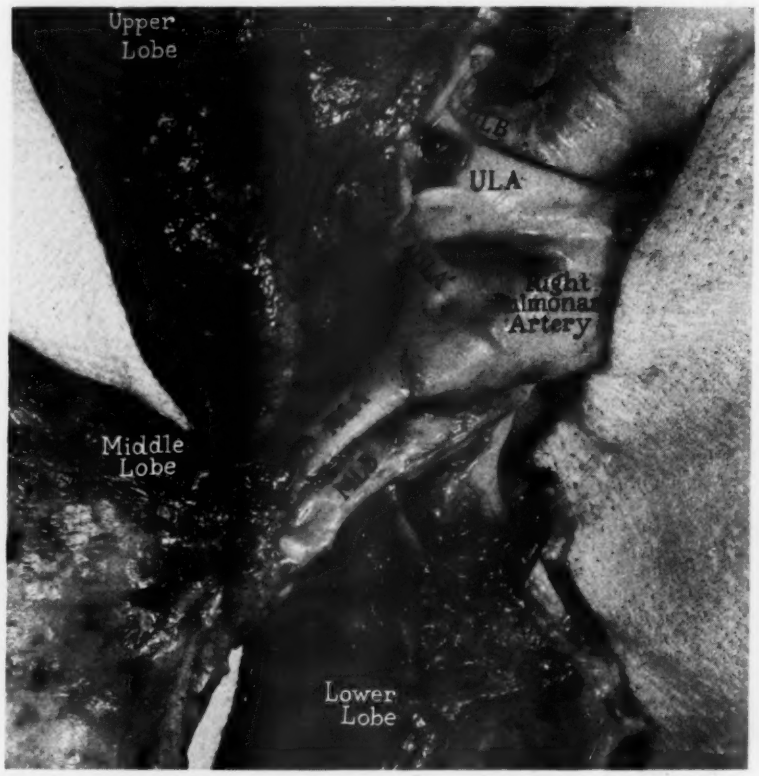

FIG. 3. B.—Dissection of right lung root. The pulmonary veins have been divided to expose the arterial distribution to the right upper and middle lobes. (ULB = upper lobe bronchus. ULA = upper lobe artery. ULA' = accessory upper lobe artery. MLA = middle lobe artery. MLB = middle lobe bronchus).

Figure 3, B. represents an illustration made after the pulmonary veins have been divided and allowed to retract. From this aspect, it is possible to see the arterial distribution to the right upper and middle lobes and the main trunk of the right pulmonary artery. It can be seen that in addition to the main arterial branch to the upper lobe, there are accessory branches which arise independently. The main arterial branches to the upper and middle lobes closely parallel the bronchi to the respective pulmonary segments. It is clear that both the arterial branches and the bronchi to these two lobes are adequate for individual dissection, division and closure.

FIG. 4. A

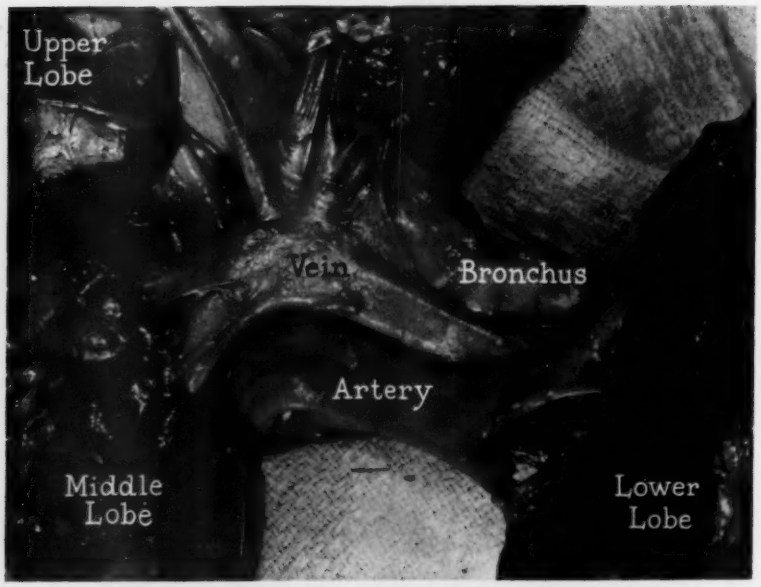

FIG. 4. A.—Dissection showing vascular supply to right lower lobe.

FIG. 4. B

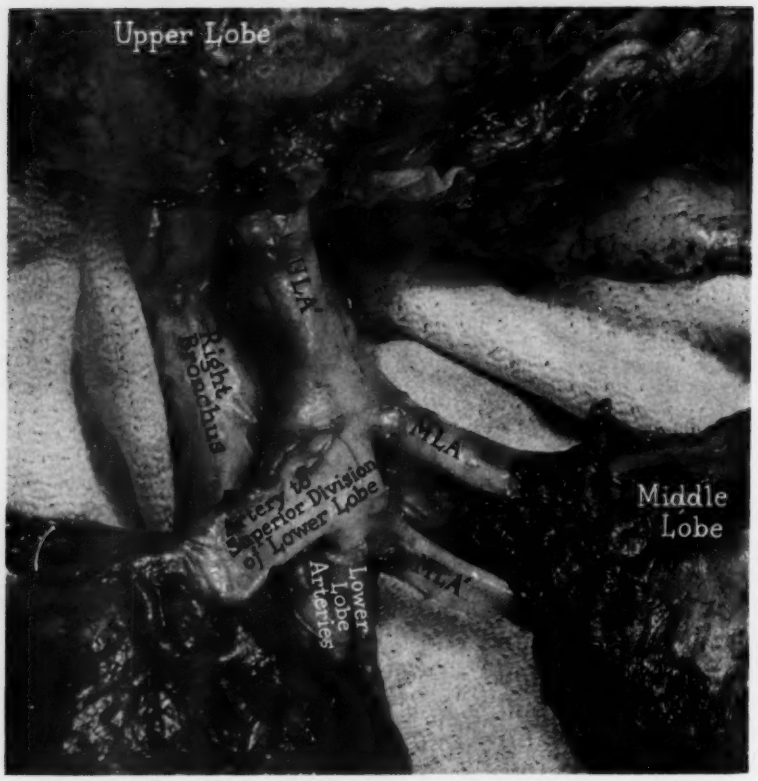

FIG. 4. B.—The vein has been removed so that arterial relationships may be emphasized. (ULA' = accessory upper lobe artery. MLA = middle lobe artery. MLA' = accessory middle lobe artery).

Figure 4, A. affords a view of the arterial and venous distribution to the right lower lobe as seen from the lateral aspect with the upper and middle lobes retracted to allow exposure. Here again, the vascular channels rather closely parallel the bronchus. However, since the venous tributary from the lower lobe has already been described as being best attacked from the anterior aspect, Figure 4, B., is presented to demonstrate the arterial distribu-

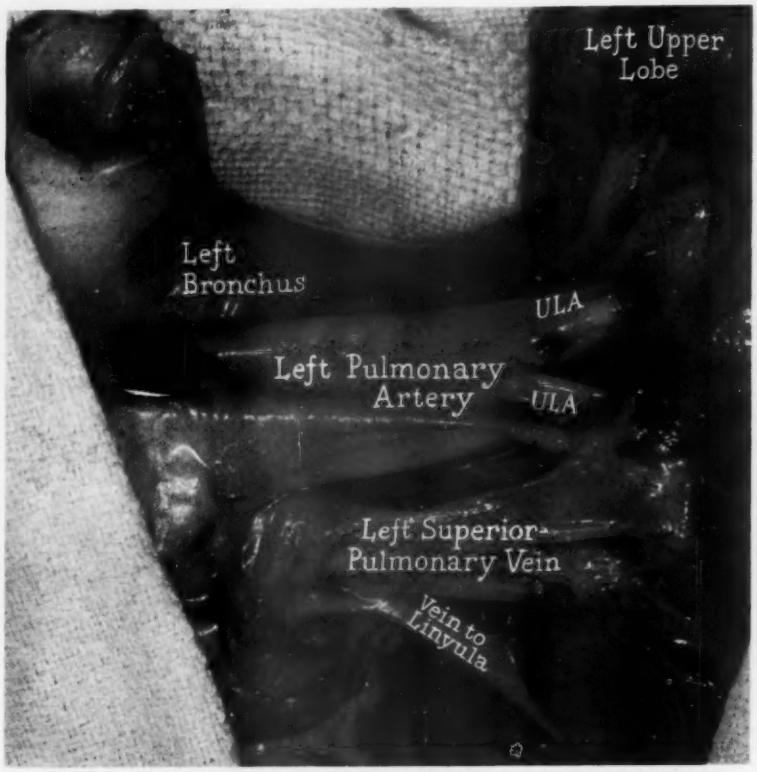

FIG. 5.—Dissection of left lung root revealing superior pulmonary vein, left pulmonary artery and left bronchus. Note upper lobe branches of pulmonary artery (ULA and ULA').

tion to the middle and lower lobes with their relationships. In this specimen there were two arterial branches to the middle lobe, each closely paralleling the bronchus. The arterial trunk continues onward to the lower lobe, but as in the case of the bronchus, the large primary subdivision is that to the apex of the lower lobe. Again as in the case of the bronchus this arterial subdivision arises at approximately the same level as do the middle lobe branches. The implications of this observation are quite clear and they are to be applied to the anatomic approach to the right lower lobe resection.

On the left side, the pulmonary veins are much like those described for the right side. Just as the middle lobe vein empties into the right superior pulmonary vein, so does the vein from the lingula empty into the left superior pulmonary vein. In repetition, let it be stated that the left inferior pulmonary

vein may be approached with certainty by pursuing the anterior surface of the left inferior pulmonary ligament.

The left pulmonary artery requires special consideration since, in our opinion, this vessel and its branches do not lend themselves to individual dissection and ligation for lobectomy as well as do the counterparts on the right side. First, the left pulmonary artery passes across the anterior aspect of the left main bronchus. Thereafter it differs from the right pulmonary artery in that it ascends over the left main bronchus to enter the fissure between the upper and lower lobes. Figure 5 demonstrates this orientation of the vessel as seen from the anterior aspect. Also, it can be

FIG. 6. A

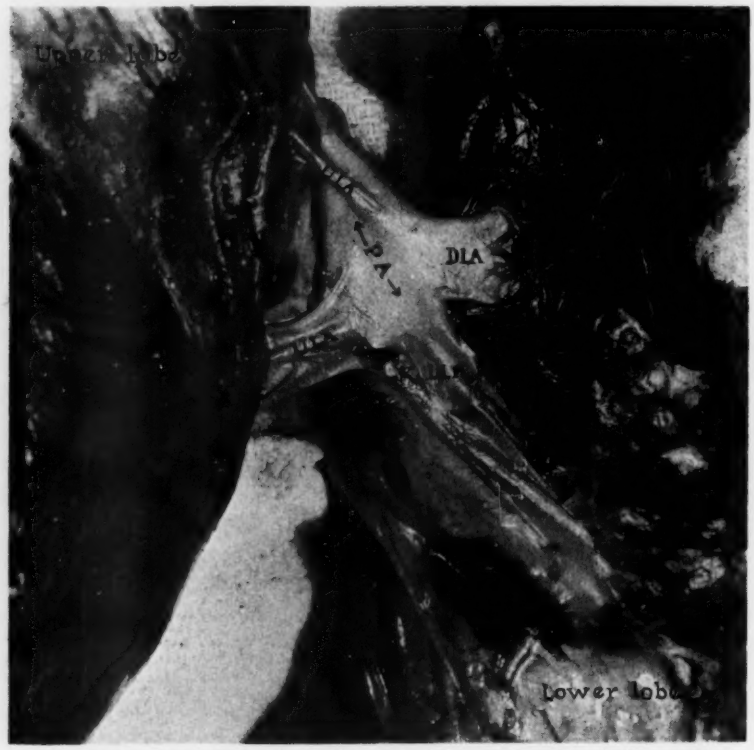

FIG. 6. A.—Dissection in the depths of the left great fissure, which revealed the arterial pattern most commonly found on the left side. The main trunk of the left pulmonary artery is shown (PA) as it courses downward and forward deep in the interlobar fissure. There are two additional branches to the upper lobe (ULA and ULA') and there are two branches to the lower lobe, the superior division or apical branch (DLA) and the terminal division of the artery (LLA). There is gross similarity to the pattern seen on the right side, the arterial division to the lingula (ULA') corresponds closely to the middle lobe artery seen on the right side. While it may be preferable to ligate the lower lobe arterial divisions separately, the main trunk may be ligated safely because there is always adequate blood supply to the upper lobe from numerous branches originating at a higher level.

seen that the artery immediately gives off branches to the upper lobe which become numerous and are short. Figure 6, A., is a view of the left pulmonary artery as it continues onward into the fissure between the upper and lower lobes. Here, additional short branches to the upper

lobe can be seen. The artery terminates in the lower lobe, but a major branch can be seen which supplies the apex of the lower lobe. Just as in the case of the relationships of the arteries to the right middle lobe and the apex of the right lower lobe, the branch to the lingula arises at about the same level as that to the apex of the left lower lobe. It should be pointed out, however, that the lingula is the recipient of a very rich blood supply from adjacent areas of the left upper lobe and thus it would not suffer from the loss of the branch shown in the illustration. Figure 6, B.,

FIG. 6. B

FIG. 6. B.—A similar dissection of another specimen. Here the pattern is complicated by more numerous arterial branches to both the left upper and lower lobes. (ULA' = accessory arterial branches to left upper lobe. LLA = terminal trunk to lower lobe. LLA' = accessory vessel to lower lobe).

illustrates a similar view of a dissection of another specimen. Here the arterial pattern is even more complicated than in Figure 6, A. There are more branches to both the upper and the lower lobes and these observations are of great important in partial lobectomy by the anatomic approach. They do not militate against successful application of the principle of individual dissection, division and closure of the structures when the left lower lobe is to be removed because, as has been pointed out, the sacrifice of one or more of the arterial branches to the upper lobe cannot be expected to compromise the pulmonary arterial circulation in the remaining left

upper lobe. We are not at all certain, however, that the anatomic approach can be applied with success to resection of the left upper lobe. There are numerous branches from the main left pulmonary artery which are short and which may be difficult to expose. It is conceivable that resection of the left upper lobe by any technic might result in obstruction of the main trunk thereby compromising the pulmonary circulation in the remaining lower lobe. Since the pulmonary artery lies in the fissure between the upper and lower lobes, it is possible, for example, that a mass ligature about the stump of the left upper lobe would include some part of the artery, thus obstructing the vessel.

The bronchial arteries provide the systemic circulatory pathways to the lungs. Observations have led to the conclusion that the position of these vessels is subject to great variation. Furthermore, they are rather small and it is to be questioned that they play any real part in maintaining viability of the lungs. In any case, these vessels have not contributed a problem during the performance of pulmonary resection. The bronchial arteries are easily dealt with when encountered. Indeed, we have been impressed by the fact that these vessels are not seen at all in many instances.

The state of the fissures between the lobes of the lungs is an important consideration. While a truly complete fissure rarely exists in man, it is often possible to extend an incomplete fissure to a point which at least approaches the required state. A study of facts about fissures has yielded the information that the middle and upper lobes are very rarely separated by a cleft of any completeness. Furthermore, the apical portions of the lower lobes are usually contiguous with adjacent areas of the upper lobes. Once the incomplete fissure has been developed between clamps, the satisfactory suturing of the divided surface of the lung has been something of a problem. To date, the use of the running lock-stitch has yielded the greatest satisfaction because it gives positive control of bleeding from the cut surface and, when tied tightly, it does not draw through to pucker the sutured portion of tissue.

The approach to the veins, arteries and bronchi is an important phase of pulmonary resection by this method. The attack upon the veins can be made through the pleural reflection which covers the anterior surface of the pulmonary hilum. An approach to the inferior pulmonary vein, using the inferior pulmonary ligament as a guide, has been described. The veins from the middle lobe and from the lingula empty into the right and the left superior pulmonary veins. However, we have seen some instances in which the vein from the middle lobe has existed as an independent third right pulmonary vein.

The approach to the main pulmonary arteries can be made in several ways. Perhaps the simplest one is made from the anterior aspect after the veins have been divided. These great vessels are in reasonably close association with their corresponding main bronchi and can usually be found without great difficulty. The chief arterial trunk to the right upper lobe can be exposed best from the anterior aspect, much as is the main trunk of

the right pulmonary artery. It is important that both the main trunk and its branch to the upper lobe be identified when the latter is to be ligated.

The arterial branches to the left upper lobe are numerous and can be found all the way from the superior aspect of the lobe root to a point well down into the interlobar fissure. The lowest branch or branches to the upper lobe are those supplying the lingular division of the lobe. The arteries to the left lower lobe are found in the depths of the interlobar fissure. In some instances these vessels stand out prominently, while in others they are completely obscured by the fusion of adjacent lung tissues of the two lobes.

The arteries supplying the right middle and lower lobes are best approached through the fissure between the upper and lower lobes. The branches to the middle lobe and to the superior division of the lower lobe commonly arise at about the same level and this point must be taken into consideration.

Once the arteries and veins have been dealt with, there is little left save the bronchus. The care with which a repair is made will appreciably influence the results in any series of cases. We are not satisfied that the best method of accomplishing this closure has been found, but its importance is so great that improved technics are constantly being sought, both by experimental means and clinical application. The pulmonary veins empty directly into the right auricle of the heart and, when very short, may require ligation of the individual tributaries rather than the main trunk. It is to be borne in mind that these structures pass through the pericardial space and that it is quite possible to open the pericardium unless care is exercised when the veins are exposed.

Preference for the individual ligation technic is based upon improved clinical results. The incidence of postoperative pleural infection is reduced, the character of the infection is more benign when it does occur. The incidence of postoperative bronchial fistula is definitely decreased, and the postoperative morbidity and mortality are favorably influenced.[4] These results are largely the consequences of a greatly improved closure of the bronchus and the elimination of a mass of strangulated, infected tissue at the stump. It is conceivable that application of individual dissection and management of the structures of the lobe root, combined with the use of chemicals of the sulfonamide group in the pleural cavity at operation, will one day make it possible to perform partial pulmonary resection with a low incidence of postoperative pleural infections.

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BRIEF COMMUNICATIONS

MEDIASTINAL GOITER

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PHILADELPHIA, PA.

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IN 1931, Cattell[1] classified lesions arising from aberrant thyroid tissue, and mentioned one extremely rare intrathoracic form in which there was no connection between the tumor and the main body of the thyroid gland. The case herewith reported is an example of this group. It should not be confused with the common intrathoracic form of goiter which develops through enlargement of nodules from the lower poles of the thyroid lobes that have become entrapped in the superior straits of the thoracic cage, so well described by Lahey.[2]

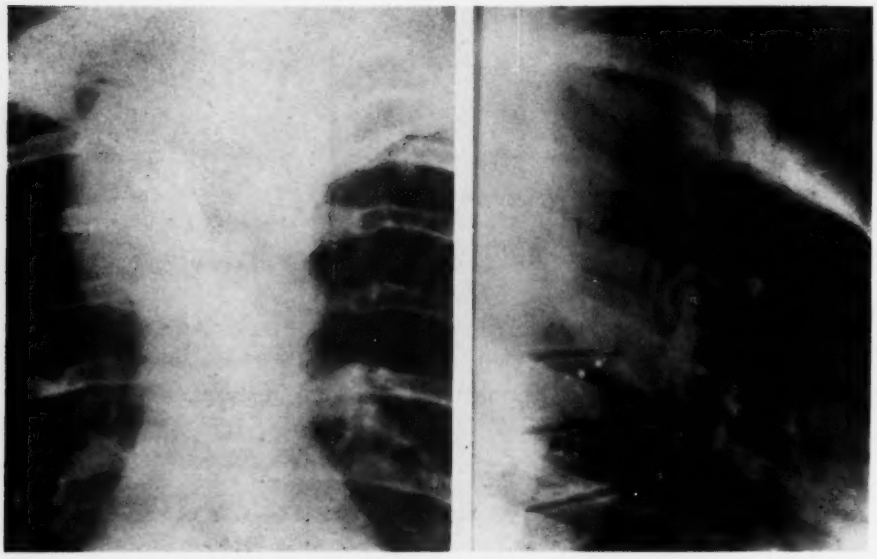
Several mediastinal tumors of thyroid origin have been reported during the past few years. Harrington[3] removed a tumor from just beneath the aortic arch, which was found to be adenocarcinoma of thyroid origin. Colloid goiter in mediastinal locations was reported by von Haberer,[4] with successful removal following division of the sternum, while Roholom's[5] case was diagnosed by aspiration biopsy and treated by roentgenotherapy. Eliason[6] and Bradshaw[7] have mentioned successful surgical extirpations of true mediastinal goiters.

Case Report.—R. J., white, female, age 19, was admitted to the Surgical Service of the Presbyterian Hospital April 10, 1939. Her previous history was of importance, for, in November, 1935, she had been admitted to this same hospital, at which time studies—not including roentgenographic visualization of the chest and neck—were made for thyroid enlargement. The essayist resected nontoxic nodular portions of each lower thyroid pole. A search revealed no nodules in the upper mediastinum or behind the trachea. The recovery was uneventful. Histologic report of the tissue was that of fetal adenoma. Several follow-up visits were made, on one of which, in 1936, a roentgenologic study of the chest and neck was made. To our discomfiture, a tumor the size of a hen's egg was discovered in the right posterior mediastinum opposite the body of the 2nd thoracic vertebra. Discussion of therapy with her parents availed nothing.

Her second admission to this hospital was caused by a severe bronchopneumonia, on February 8, 1939. She made a good recovery, without complications. Roentgenologic studies of the chest during this admission showed an increase in the size of the tumor. It was reported as a smooth, rounded tumor in the right posterior mediastinum, extending from the level of the bodies of the 2nd to 5th thoracic vertebrae (Fig. 1A and B).

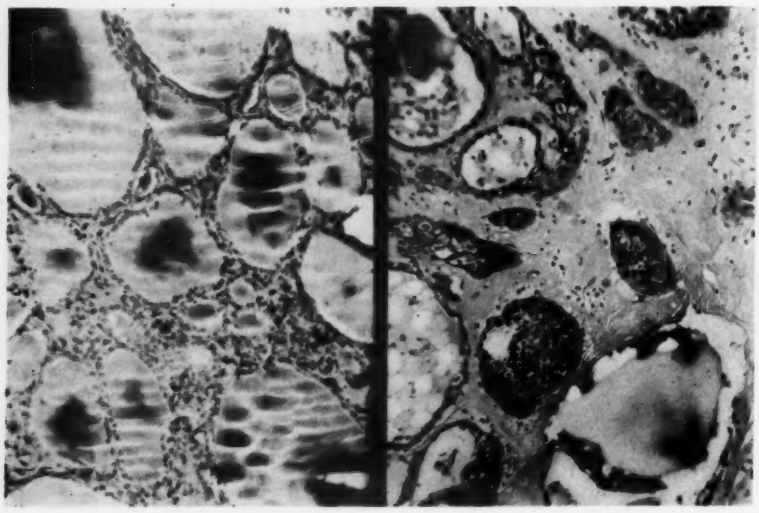
After somewhat less than two months convalescence, she was readmitted to the hospital at the insistence of her family, who had been informed of the increase in size of the tumor, which, however had caused no symptoms. Because of the patient's approaching marriage her parents not only listened to advice but readily acquiesced to surgical management of the lesion.

* Read before The Philadelphia Academy of Surgery, February 3, 1941.



(A) (B)

FIG. 1.—(A) Anteroposterior roentgenogram showing the extent of the tumor mass. (B) Lateral roentgenogram showing the tumor mass and its relation to the air-filled trachea and the esophagus containing barium mixture.



(A) (B)

FIG. 2.—(A) Photomicrograph showing typical acinar formation of a nontoxic nodular goiter. (B) Photomicrograph showing acinar areas containing colloid, others containing sheets of epithelial cells and, also, an excessive amount of fibrous tissue between the acini.

Pertinent studies of blood chemistry, blood count, serology, and urinalysis showed normal figures. The basal metabolic rate was within normal range. Vocal cords approximated normally. Barium visualization of the esophagus revealed no compression, while roentgenograms of the chest confirmed earlier findings and location of the tumor. Physical examination revealed no chest signs, and a local palpation of the thyroid area showed freedom from recurrence in either lobe.

Operation.—April 15, 1939: Under cyclopropane intratracheal anesthesia, the mediastinum was entered through a right posterior incision. A curving incision about one inch medial to the vertebral border of the scapula was made through skin, subcutaneous tissue and muscle down to the ribs. A section of the 5th rib was removed close to the spine, and its bed was carefully excised, without piercing the pleura. The parietal pleura

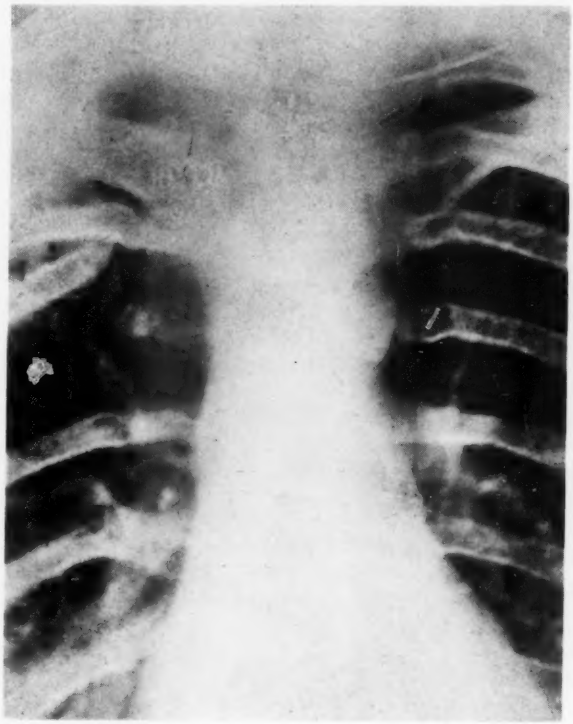


FIG. 3.—Follow-up anteroposterior roentgenogram showing area previously occupied by the tumor.

was mobilized from this point upwards and downwards, and the 6th, 4th and 3rd ribs were rapidly sectioned. Mobilization of the pleura was continued until the tumor was adequately exposed. It was smooth, rounded, and cystic in consistency, red-brown in color, and between three and four inches in diameter. It rested against the right anterior aspect of the bodies of the thoracic vertebrae from the lower border of the 2nd thoracic to the upper border of the 6th thoracic. The lung hilus was anterior and below. Internal to the tumor were the trachea and esophagus. In this latter organ an esophagoscope had been placed as a guide. The great vessels and brachial plexus were above and anterior. There was no palpable connection with the thyroid gland in the neck nor with any structure in the mediastinum, other than the tumor-bed. The capsule of the tumor was incised and it was enucleated with ease. The capsule was closed by interrupted catgut suture, which controlled bleeding. The tissue removed was cystic and nodular, and of the

consistency of adenomatous thyroid tissue. A cigarette drain was brought down to the tumor bed through an opening in the old collar incision. This unusual placement was an error—though it was done to save the prospective bride a possible draining back-sinus. The rib ends were approximated with chromic catgut, and the incision closed in layers, without drainage. A transfusion of 500 cc. of citrated blood was begun shortly before conclusion of the operation through a cannula which had been placed in a foot vein before beginning the operation.

Immediate convalescence was uneventful until the 5th day, at which time the catheter which had been placed through the cigarette drain to the tumor-bed ceased to respond to suction. On the 10th day (April 25) a septic temperature developed, and roentgenograms of the chest showed a fluid level, extrapleural in location. Culture of this fluid showed hemolytic *Staphylococcus aureus, Streptococcus viridans* and *Bacillus proteus.* Reestablishment of drainage caused abatement of the fever. Time was lost by improper use of chemotherapy (i. e., without proper drainage) so that a persistent, variable increase in temperature persisted. On the 43rd day the posterior incision was opened and thick yellow pus was evacuated, which showed the previously mentioned bacterial flora. The septic temperature ceased and the wound healed rapidly. Seventeen days later, roentgenograms of the chest showed almost complete disappearance of mediastinal and retropleural densities. The patient was discharged June 22, 1939.

Pathologic Report.—Dr. Philip Custer. Tumor mass seven centimeters in diameter, weighing 105 grams; multilocular, thin capsule filled with cystic areas each containing red-brown material. Microscopic examination revealed thyroid tissue in irregular acinus formations, except in one area where there was fibrous tissue, and in a few other areas solid sheets of epithelial tissue lined the cystic spaces. It is not possible to tell whether this is metaplasia, or that it is a mixed tumor made up almost exclusively of thyroid tissue (Fig. 2A and B). *Pathologic Diagnosis:* Aberrant thyroid.

Follow-Up Visits. The patient made several follow-up visits. Roentgenograms of the chest, taken September 1, 1939, showed thickened pleura and malposition of the fragments of the third rib (Fig. 3). This deformity was due to the poor selection of material to oppose the fragments. The patient was married September 15, 1939. Her last follow-up visit made in the spring of 1940. She had gained considerable weight and strength, and was well in all respects.

SUMMARY

The successful removal of a posterior mediastinal, nontoxic nodular goiter by extrapleural mediastinotomy, and complicated by secondary extrapleural hemolytic *Staphylococcus aureus* and *Streptococcus viridans* infection is recorded.

Acknowledgement is made to Dr. William Bates, Chief of Surgical Service in the Presbyterian Hospital, for his aid and advice in this case.

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BOOK REVIEW

THE SURGERY OF PANCREATIC TUMORS. By Alexander Brunschwig, M.D., St. Louis, Mo.: C. V. Mosby Co., 1942.

IN THESE HECTIC DAYS of global warfare one must admire the courage and faith of the author, and of the publisher, of a monograph not related to any phase of our present emergency. But in reviewing the comprehensive and authoritative volume by Dr. Alexander Brunschwig on the "Surgery of Pancreatic Tumors," we would emphasize the fact that the author has been studying the subject and contributing to it for many years. The motive and purpose of the monograph is best presented in Brunschwig's introduction: "A medical treatise may be written to cover a large and well known field and thus serve as a review, and for instructional progress; or it may deal with controversial subjects and thus afford its author the opportunity to state his own views; or it may deal with relatively few facts in a newly developing field and serve to stimulate further work in this field. It is in this latter spirit that this treatise was written. A summary of most of the recorded experiences in the surgery of all types of pancreatic tumors is attempted, with the hope that it might contribute in some small measure to the maintenance of interest in this branch of operative surgery, especially in regard to malignant growths."

The first chapter, reviewing the history of the experimental pathology and surgery of the pancreas and the early attempts to deal with malignancy of the organ, shows a thorough study of the literature. Especially interesting are the references to the early contributions of Brunner and of Graaf. This historical review is, in itself, a unique contribution. Chapters II, III and IV take up in detail the anatomy and physiology and the experimental surgery, respectively, and add very materially to the value of the book as a monograph on the pancreas, aside from the main theme of neoplasms of that organ.

The chapter on diagnosis of pancreatic lesions and diagnostic procedures is followed by one on preoperative and postoperative care. Both of these are comprehensive, give all the newer advances, and do not repeat out-of-date and useless tests and methods.

Three chapters deal with pancreatic cysts, followed by one devoted to cystadenomata and cystadenocarcinomata. Lesions of the ampulla of Vater, with a discussion of differential diagnoses, are considered at length.

The next seven chapters discuss the most recent advances in the surgery of the pancreas, five of these on carcinoma of the acinar and duct epithelium, followed by two chapters on islet cell tumors. These are original comprehensive contributions to new fields of surgery and in themselves could be given the title of the monograph. These pages deal with new data in a newly developing surgery and serve to stimulate further work in this field. They constitute a summary of most of the recorded and much of the unpublished experiences in the surgery of all types of pancreatic tumors.

From this brief review, it is obvious that Dr. Brunschwig's monograph is as much a treatise on the pancreas as on the tumors of the pancreas. But the reviewer would emphasize again the original contributions to the newer surgery of the tumors of the pancreas that Dr. Brunschwig has made in this book.

ALLEN O. WHIPPLE, M. D.

BOOKS RECEIVED

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